

SOLITARY LESION IN PONTO-MESENCEPHALIC AREA RELATED SECONDARY MANIA: A CASE REPORT

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INTRODUCTION

Bipolar I disorder begins frequently between the ages 15-24 and differs from other mood disorders (MD) in that at least a manic or mixed episode is needed for the diagnosis. An older beginning of bipolar I disorder should suggest another primary cause such as a general medical condition, organic causes or drug use. The most common causes of secondary mania are neurological causes. Focal brain lesions are most common causes of secondary mania and the most important causes of focal brain lesions are multiple sclerosis, Parkinson's disease, epilepsies, various causes of dementia, brain aneurisms, brain tumors, traumatic brain injuries, cerebrovascular lesions (Jacobson et al. 2007).

We present a case of mania probably caused by a lacunar infarct in the pontomesencephalic area.

CASE REPORT

The patient's family members brought him to our clinic. He was 62 years old. On admission, the patient had euphoria, irritable mood and hyperactivity. We observed increased self-confidence, distinct compressive speaking, flight of ideas, logorrhea, no tiredness though decreased sleeping time and increased movement, and grandiose delusions, for instance that he was a great businessman and could achieve every job 10 days before being brought to our clinic, he suddenly fainted, lost consciousness and he was taken to an emergency clinic. His blood pressure was found to be high. When admitted to the emergency department, systolic blood pressure was 220 mmHg and diastolic blood pressure was 140 mmHg. He was not on any regular antihypertensive medication and 50 mg of captopril was given orally. The blood pressure level decreased to 130 mmHg systolic and 80 mmHg diastolic. Hypertensive encephalopathy was diagnosed at that point. His consciousness returned, his neurological and other physical examination were normal. After 12 hours follow-up, he was discharged with oral 20 mg olmesartan and 12.5 mg of hydrochlorothiazide therapy. Psychiatric examination was not performed at the emergency department. His mental status was normal at discharge. 2-3 days after

this fainting episode, he began to be too active with continuous walking around at home, sleepless with only 1-2 hour or no sleep per day, too cheerful. He was spending too much money, dreaming about big jobs, loaning great amounts of money for new and impossible concerns, getting financial troubles and becoming aggressive and threatening when he felt that the family members were trying to hinder him.

His medical history revealed that he had been smoking for 35 years in the past. His height was 1.72 m and weight was 97 pounds. Psychiatric history did not include previous episodes of mania or depression. However, he had no history of head trauma. Results of neurological examination, general physical examination, and laboratory investigation, including blood chemistry, thyroid function test, FSH, LH levels, were normal. The patient then had an electroencephalogram (EEG) and the result of this test was within normal limits. His routine daily blood pressure examinations revealed high blood pressure levels. He was taking antihypertensive medication but when the patient was brought to our attention, arterial blood pressure level was 150 mmHg systolic and 90 mmHg diastolic. 25 mg of captopril was given orally. Blood pressure level decreased to 130 mmHg systolic and 80 mmHg diastolic.

Magnetic Resonance Imaging (MRI) examination was also performed. MRI revealed a lacunar infarct in the pontomesencephalic area of the left hemisphere (Figure 1 and Figure 2). MRI imaging did not reveal any other pathological finding.

After neurological consultation, 300 mg/day acetyl salicylic acid (ASA) was started. Aripiprazole was found a suitable choice of treatment for the manic symptoms, since the patient was overweight and of a relatively older age. Aripiprazole 5 mg/day was started. After 10 days the dose was increased to 10 mg aripiprazole/day since the improvement was not complete. Manic symptoms regressed in 2 weeks and improved completely after the end of the 3 week treatment. The patient was discharged with 10 mg/day aripiprazole. 2 weeks, 1 month, 2 months follow-ups after discharge revealed no signs of mania and 10 mg/day aripiprazole continuation treatment was advised. On follow-up, he was taking 300 mg of ASA and olmesartan hydrochlorothiazide 20/12.5 mg beside psychiatric therapy. Blood pressure level was regular.

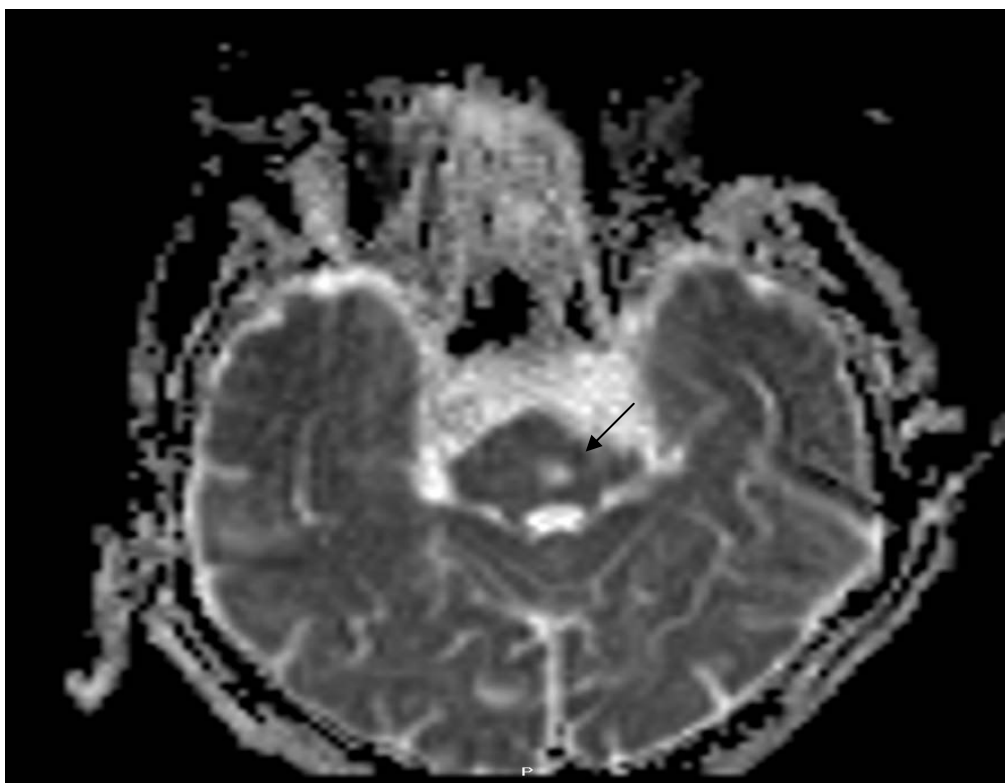


Figure 1. MRI diffusion image

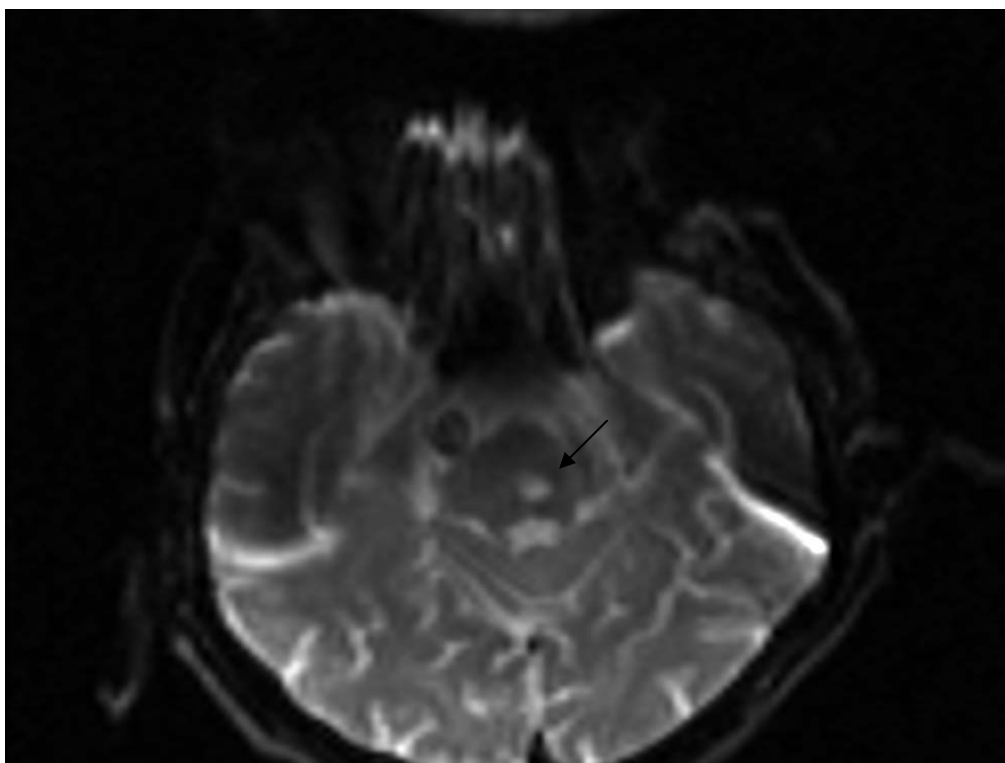


Figure 2. T2 image

DISCUSSION

The available results suggest that certain factors including smoking, advanced age and hypertension increase the potential for occurrence for stroke (Goldstein et al. 2011). The patient had risk factors for

cerebrovascular stroke including smoking, advanced age and hypertension.

Secondary mania is a rare condition (Clark et al. 2001). It can have many causes and the most probable of them is thought to be focal lesions of brain (Jacobson et al. 2007). Many different anatomic locations which

could be related to secondary mania and bipolar disorder have been determined in some studies. The frontotemporal turn abnormalities in these patients were pointed out to be possibly important in the development of this disorder (Tamashiro et al. 2008). In some studies various localizations of lesions including basal ganglia containing the right caudate nucleus and especially the temporobasal region of the limbic system were defined (Starkstein et al. 1990). Solitary lesions of the temporal lobe were also reported to be possibly related to the secondary manic syndrome (Mendez 2000, Starkstein et al. 1988). While right frontoinferolateral and right temporobasal lesions were seen to be related with pure manic syndrome (Jorge et al. 1993, Joseph 1999), right caudate and thalamic lesions were seen to be related with bipolar disorder characterized by recurrent manic and depressive periods (Robinson et al. 1988, Starkstein et al. 1987). Cerebellar-pontine lesions have also been implicated because these areas are connected with frontal- and posterior parietal-subcortical-thalamic circuitry via inputs to the thalamus (Amino et al. 2001, Nagao 2004). Nishio et al. (2007) performed stereotaxic lesion localization on magnetic resonance imaging (MRI) and statistical analyses of regional cerebral blood flow (rCBF) in a patient who developed frontal-lobe syndrome and psychotic symptoms after infarction in the pontomesencephalic junction. The results suggested that the patient's symptoms were ascribable to disruption of the ascending dopaminergic projections to the frontal-subcortical circuit components. They found rCBF reduction also in the left temporo-parietal cortex at the first SPECT session. The projections arising from the rostral brainstem dopaminergic nuclei dominate in the frontal cortices but also terminate in the posterior cortical regions (Nishio et al. 2007).

Clinical observations and neuroimaging examination suggest that our patient's frontal-lobe-like and manic symptoms were ascribable to damage to the rostral brainstem dopaminergic nuclei and its projections to the frontal-subcortical circuit components. This infarct region might have caused the manic syndrome by affecting the same pathways.

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Conflict of interest : None to declare.

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