Manic Episodes Possibly Secondary to Pediatric Ischemic Stroke

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Background: Post-stroke mania has rarely been reported, and the lesions of most reported cases have been involved in the non-dominant hemisphere of the brain. We reported a case of mania probably secondary to stroke with a lesion in the dominant hemisphere. Case Report: A 22-year-old, right-handed male patient had a history of pediatric stroke involved in the left middle cerebral artery territory of the brain. He became depressed right after the stroke, and five years later he developed a manic episode. He received lithium and risperidone with satisfactory response. Conclusion: The brain lesion other than laterality may play an important role on the etiology of mania. The research on secondary mania after the stroke is needed to explore the relationship between mood and brain functional region.

Keywords: secondary mania, stroke, left frontal lesion, pediatric condition


Introduction

Numerous neuropsychiatric disturbances which occur following the cerebrovascular diseases, include anxiety, depression, mania and psychosis. Among them, mood disorder secondary to stroke has been widely studied and reported. Depression is often observed after the brain lesion. It is thought that depression in a right handed patient is associated with left frontal lesion of the brain [1]. But post-stroke mania has rarely been reported. One study identified only three cases among more than 300 stroke patients [2]. Those patients have typical manic symptoms, and have no difference between those with and without brain injury. Most reported cases have been involved in the right, non-dominant hemisphere of the brain in the elderly [3]. Here we report a case of manic patient who probably had secondary mania after a pediatric stroke with a lesion in the dominant hemisphere of the brain.

Case Report

A 14-year-old, right-handed, previously healthy adolescent patient without family history of mood disorder and other vascular disease, had a sudden onset of consciousness disturbance in December 2000. The result of neurological exam-
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In the patient's presentation, weakness on the right side of the body was noted along with dysarthria. The finding of computed tomographic (CT) study revealed cerebral infarction involving the left basal ganglion, the head of the left caudate nucleus, left putamen and part of the left corona radiata. The cause of the infarction was suspected to be related to cerebral vasculitis with slightly lowered serum level of antithrombin-III factor, which was returned to normal range 18 days later. He did not have any evidence of systemic vasculitis or connective tissue disease.

He was discharged after a 27-day hospitalization with mild reduction in the muscle power and speech fluency. He was seen regularly once a month in the neurology clinic for one year, and took aspirin and piracetam. His academic performance was worsened significantly after the stroke.

In May 2007, 6.5 years after the stroke, he showed decreased need in sleep, racing thoughts, irritability, hyperactivity, hypersexuality, inflated self-esteem, grandiosity and persecutory delusion. In physical examination, he did not have any new finding of neurological deficits. He had normal values of laboratory tests including complete blood count, urinanalysis and biochemical screening. The finding of the brain CT did not reveal any new neuroimaging changes. He started to receive daily lithium 900 mg and risperidone 4.5 mg as well as aspirin and piracetam. He became much improved under medical treatment and was discharged 35 days after admission.

The dosage of risperidone was tapered off to 1.5 mg because of stable psychiatric condition. During the follow-up course in outpatient clinic, his dosage of risperidone was gradually tapered off and discontinued in December 2007. The serum lithium levels were all within therapeutic range.

In April 2008, the patient interrupted taking medications occasionally, and totally discontinued them three days before his second hospitalization. Thus, he developed another manic episode with presentation of decreased need of sleep, hyperactivity, hypertalkativeness, irritability, aggressive behaviors, and persecutory delusion. Thus, he was hospitalized again. We resumed his previous medications, lithium 1200 mg per day and risperidone 3 mg per day. After the treatment for about one month, his manic symptoms were under control and then he was discharged home.

**Discussion**

This patient's presentation of decreased need of sleep, racing thoughts, irritability, hyperactivity, hypersexuality, inflated self-esteem, grandiosity and persecutory delusion, met the DSM-IV-TR criteria for manic episodes, and also met the criteria recommended for vascular mania by Staffans and Krishnan [4]. But the recent studies on vascular mania have been focused on late-age onset group, and the prevalence of pediatric stroke is low. Although a growing evidence exists to use 50 years as the cut-off age for having vascular mania, attention should be paid to the neuroimaging changes and the neuropsychological changes as in our patient.

Among the reported cases, some authors have described cases with simultaneous onset of mania or immediately after stroke [5], some have reported an average of six months [2], and others also have reported as long as two years after the
stroke [5]. The interval from stroke to the onset of mania is variable according to current research information. Our patient developed mania 6.5 years after the stroke. We suggest that a lesion may not be able to cause secondary mania by itself. There may also be predisposing variables other than the brain lesion contributing to the final presentation of manic symptoms. For instance, psychosocial variables and life stressors may be important as well.

Post-stroke depression is correlated with lesions in the left hemisphere, and the left frontal lobe and the basal ganglia are the most related regions [6]. Our patient had the main brain lesion involved in the left basal ganglion, the head of the left caudate nucleus, left putamen and part of the left corona radiate. He developed dysthymia developed soon after the stroke, and it may be related to lesion in left hemisphere.

Lesions in the right hemisphere of the brain appear to be most often associated with post-stroke mania, especially involving in the right orbitofrontal, basotemporal cortex or areas related to the limbic system [2]. A study suggests that disruption of right subcortical mechanisms may be important in bipolar disorder, and disruption of right cortical mechanisms may mediate a unipolar mania [7]. One study showed a case with right-sided infarction, and post-stroke SPECT finding showed a pattern of left orbitofrontal hyperperfusion with extensive right frontal hypoperfusion, suggesting that a functional imbalance between right and left orbitofrontal cortices may be important in mania [8].

Our patient was successfully treated with lithium and risperidone in the acute manic phase, and took lithium alone as maintenance therapy. So far, no systemic studies exist on the treatment of mania after a cerebrovascular disease, and the existing case reports reflect various treatments and responses. The underlying organic factors also complicate the treatment and response. Lithium has been the most commonly used medication for secondary mania, and anticonvulsants and antipsychotic agents are the second most used [9]. Although the pharmacologic treatment of secondary mania is variable, it typically follows the same regimens used for primary mania.

Mania secondary to left hemisphere lesion of the brain is a less common finding although there are still some reported cases [10, 11]. Our patient’s mania was probably following a dominant hemispheric stroke. It may appear to be overlapped between primary and secondary mania. We suggest that research are needed to explore the relationship between mood and brain location/function.

References


