

Case report

Bipolar disorder following a stroke involving the left hemisphere

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Objective: Post-stroke mania has rarely been noted and researched, and reported cases have mostly involved the non-dominant hemisphere. In this paper, we report a case of bipolar disorder secondary to a stroke over the dominant hemisphere.

Clinical picture: A 48-year-old, right-handed man had a cerebral infarct over the left temporal region. He became depressed after the stroke, and 4 months later developed a manic episode.

Treatment: The patient was treated with haloperidol and lorazepam.

Outcome: The symptoms subsided within 2 months after treatment.

Conclusions: It is premature to consider mania to be a syndrome of the right, or non-dominant, hemisphere. We suggest that further study focusing on specific anatomical regions, rather than laterality, will help to elucidate the inter-relationship between mood and brain function.

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Secondary mania refers to the occurrence of symptoms which meet the diagnostic criteria for mania produced by neurological, metabolic or toxic disorder [1]. Focal brain lesions, including haemorrhage and infarction, have been associated with secondary mania [2,3]. The frequent occurrences of depression after a stroke have been documented in the literature [4]. In contrast, mania after stroke has rarely been noted [2,5].

Dunne *et al.* [6] identified only three cases of post-stroke mania among 661 stroke patients. Starkstein *et al.* [7] also identified three cases in a series of 700

consecutive stroke patients. Most cases reported had a lesion in the non-dominant hemisphere [2,3]. Only four cases of post-stroke mania with left hemisphere lesion have been reported [7–10]. Among them, two cases presented both depressive and manic episodes [10].

We report another case of bipolar disorder secondary to a stroke involving the left hemisphere.

Case report

A 48-year-old, right-handed, previously healthy man had a sudden onset of speech difficulty in June 1993. Sensory aphasia was discovered by neurological examination, and a computerised tomography scan of the brain showed a suspicious low density lesion over the left posterior temporal region. A magnetic resonance image of the brain 4 days later showed a low signal on T1-weighted (Fig. 1a) and a high signal on T2-weighted (Fig. 1b) images over the left temporal region, consistent with a recent infarction.

An electroencephalogram revealed continuous polymorphic delta activity over the left temporal

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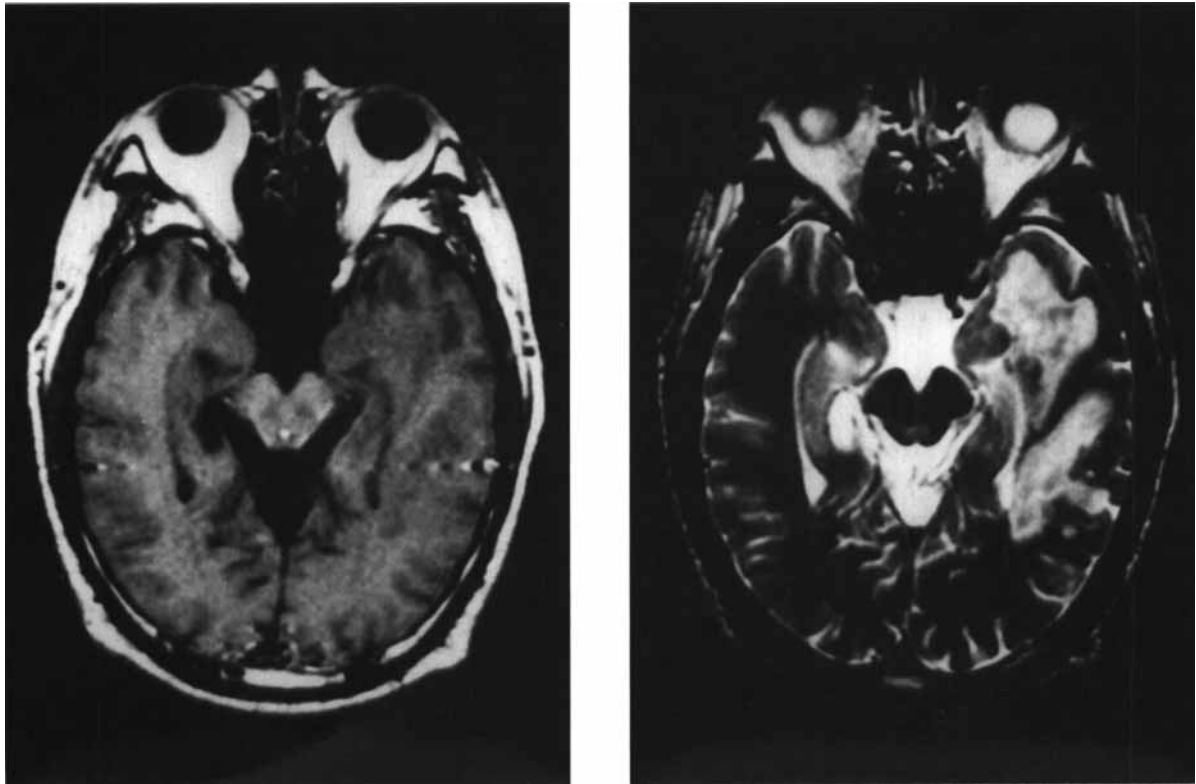


Figure 1. Magnetic resonance image of the patient's brain showing a low signal on (a) T1-weighted and high signal lesion and (b) T2-weighted images in the left temporal region, compatible with recent infarction.

region. The patient became depressed after the stroke. Clinical features included depressed mood, sleep disturbance, inappropriate guilt, poor appetite and the idea of death. Benzodiazepines, such as lorazepam and estazolam, were the only drugs prescribed by neurologists. The depressive symptoms subsided within 3 months. Sensory aphasia also gradually improved. There were no major neurological sequelae except mild impairment of abstract thinking and comprehension. The patient was able to do domestic work and returned to his job as an office worker with fair proficiency.

Four months later, the patient experienced diminished sleep, became increasingly irritable, hyperactive, hypertalkative and elated with grandiose ideation. Sent to our emergency room, he was given a physical examination which yielded essentially unremarkable results, except an extensor plantar response on the right side. His blood pressure was 150/90 mmHg with a regular heart rate 88 bpm. He had neither a personal nor a familial history of psy-

chiatric illness, nor a history of symptoms suggesting recurrent depression; and neither a history of episodic change of energy, nor that of unusual periods of expansive, erratic or provocative behaviour. He had a history of hypertension for 12 years and non-insulin dependent diabetes mellitus for 4 years. Both were under regular treatment.

Throughout his mental status examination, the patient was fully alert and well-oriented. His thoughts, with grandiose and religious content, flowed rapidly from idea to idea. His mood was euphoric and elated. He exhibited loud, abusive and pressured speech. His digit span was seven forward and four backward. He performed calculation normally and could recall three objects after 3 minutes without difficulty. No hallucination or delusion could be detected.

Laboratory data, including complete blood count, urinalysis and biochemical screening, all were within normal limits, except fasting blood sugar (147 mg%). Computerised tomography scan of the brain showed

an old infarction over the left posterior temporal area. No additional lesion was noted.

During hospitalisation, the patient was intrusive, hyperactive and disinhibited. He felt full of energy, wandered around and talked to others all day long, and stayed awake all the night. Occasional abortive episodes of tearfulness were also noted. He was treated with haloperidol 8 mg per day, trihexyphenidyl 1 mg t.i.d., lorazepam 0.5 mg t.i.d., and estazolam 2 mg before sleep. All symptoms gradually improved. He was discharged 2 weeks later. Psychotropics were discontinued 2 months after discharge.

Discussion

This patient met the DSM-III-R criteria for organic mood disorder [11] and Krauthammer and Klerman's criteria for secondary mania [1]. Although Krauthammer and Klerman [1] and Stasiak and Zetin [12] considered 'secondary mania' should be reserved for cases without personal or family history, some other authors suggested prior vulnerability may predispose to organic mood disorders [13]. In this case, the onset followed a stroke without a personal or familial history of mood disorder. It is certainly suggestive of a causal relationship.

Among the reported cases, mania may occur immediately after stroke or may be delayed for up to 2 years [7]. In some cases, recurrent episodes of depression and mania were quite similar to an idiopathic bipolar disorder [10,12–15]. Starkstein *et al.* [16] indicated that patients with right subcortical lesions tend to develop bipolar disorder; whereas patients with right cortical lesions tend to develop unipolar mania. Pappata *et al.* [17] suggested that

subcortical lesions may produce hypometabolic effect in both ipsilateral and contralateral brain areas, which may lead to bipolar mood changes. But in the present case, no subcortical lesion was noted.

A conventional hypothesis says that the organisation of mood in individuals may be lateralised to the right or non-dominant hemisphere [7,13,18–20]. Our case is an example of the less common findings. Not only was the patient right-handed, but he also had transient post-stroke aphasia. This is the fifth case reported in the literature in which secondary mania followed a stroke in the dominant hemisphere [7–10], and it is the third case in which both depressive and manic episodes occurred (Table 1).

Cummings [3] reviewed the reported cases and concluded that most lesions associated with secondary mania involved the diencephalic region. Robinson *et al.* [2] indicated that the patients with secondary mania had lesions involving mainly the right orbitofrontal, basotemporal cortex or areas related to the limbic system. Bogousslavsky *et al.* [21] suggested that an ipsilateral frontal dysfunction caused by deafferentation due to a right thalamus lesion may produce a manic-like disinhibition syndrome. Other than laterality, they pointed out some specific regions which may be responsible for the illness.

In a review article on post-stroke depression, Starkstein and Robinson [5] contended that the lateralised effect of post-stroke depression was restricted to left-sided basal ganglia and left frontal lesions. It is probable that the lateralised effect of post-stroke mania is also restricted to right-sided diencephalon- and limbic-associated lesions.

The authors suggest that it is premature to consider mania to be a syndrome of the right, or non-domi-

Table 1. Reported cases of mania following left hemisphere stroke

Authors	Handed	Type of lesion	Location
Herlihy and Herlihy [8]	Unknown	Haemorrhage	Left frontotemporal
Jampala and Abrams [9]	Right	Haemorrhage	Left frontotemporal
Starkstein <i>et al.</i> [7]*	Right	Infarction	Left frontal
Tureki <i>et al.</i> [10]*	Right	Unknown	Left basal ganglia
Present case*	Right	Infarction	Left temporal

*Presenting both depressive and manic episodes

nant, hemisphere. Further study focusing on specific anatomical regions, rather than laterality, will help to elucidate the inter-relationship between mood and brain function.

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