

Clinical difficulties in post-stroke mania

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Dear Editor,

A 52-year-old man presented to our outpatient psychiatry department due to elevated mood, reduced need of sleep, elevated energy, disinhibition and grandiose delusions during the last two months.

This patient is married, has two children and lives with his family. He is partially dependent in activities of daily living (walking with help).

Of relevance, he had a left lenticulo-capsular hematoma and underwent decompressive craniectomy two years before the onset of psychiatric symptoms. After stroke, the patient presented sequelae of right hemiparesis, motor aphasia and late-onset seizures, without evidence of cognitive impairment (normal Mini Mental State Examination score).

Past medical history included hypertension, dyslipidemia and hyperuricemia. No previous history of any psychiatric disorder, including mood or substance use disorders, could be evidenced.

When psychiatric symptoms occurred, he was medicated with levetiracetam for the seizures. The first option of his neurologist was excluded medical conditions that could justify these psychiatry symptoms. Then, it was decided to change the antiepileptic drug and the patient stopped levetiracetam and started lamotrigine, without psychiatric improvement. He started quetiapine up 400 mg/day and after olanzapine up 20 mg/day was associated, both without psychiatry improvement. Then, the patient started pimozide up 8 mg/day with clinical improvement and after 6 months of treatment it was progressively discontinued, without psychopathological decompensation.

At 6-months follow-up, he was not medicated with any psychiatric drug and had no recurrence of psychiatric symptoms.

This patient met the Krauthammer and Klerman's criteria for secondary mania¹.

This case is consistent with the typical stroke-related mania patient described in the literature: male, presence of vascular risk factors (hypertension, dyslipidemia and hyperuricemia), absence of psychiatric history and subcortical atrophy. However, there are some peculiarities, particularly the presence of a left stroke. It was been found a significant correlation between post-stroke mania and right hemispheric lesions^{2,3}. However, there are an increasing number of reports of post-stroke mania in people with left hemisphere involvement^{2,4}. In fact, left lesions can also lead a dysfunction in the ventral limbic circuit that is a key to mood regulation and social behavior⁴.

According to the literature, the prevalence, incidence and course of post-stroke mania are not known. Probably these cases were underdiagnosed in the stroke context specially in patients with major symptoms like afasia¹.

Post-stroke mania should be considered in any maniac patient who presents concomitant neurological deficits and is older than expected for the onset of primary mania⁴. In our patient, the maniac episode occurred two years after the stroke. We suspected that these psychiatric symptoms are related to the previous stroke, because it was excluded other possible causes. The presence of physical post-stroke sequelae, particularly hemiparesis and aphasia and the treatment with levetiracetam for vascular epilepsy delayed the diagnosis of stroke-related mania.

There are no official guidelines on how to manage stroke-related mania. This patient was treated with typical antipsychotic that has low impact on the seizure threshold and there was a good psychiatric response.

Our case illustrates the clinical challenges in the evaluation of psychiatric symptoms presenting in post-stroke follow up.

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Conflicts of interest

The authors do not have any conflicts of interest.

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