

Post-stroke emotional incontinence or bipolar disorder?

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Introduction: Post-stroke emotional incontinence and bipolar disorder are two disorders that involve the dysfunction of brain structures responsible for emotional regulation. The objective of this work is to study the links between these disorders through a clinical case.

Case report: We present the case of a 43-year-old man without previous psychiatric history who experienced emotional incontinence after cerebrovascular events. He reacted promptly to selective serotonin reuptake inhibitor treatment. However, he experienced his first episode of hypomania after 6 months of antidepressant therapy. Adjunctive therapy with valproic acid and low-dose paroxetine was eventually added, resulting in complete improvement of both emotional incontinence and hypomania after 4 additional months of treatment.

Conclusion: The clinician should carefully explore any history of premorbid bipolar disorder, personality disorder characterized by mood instability, and family history of bipolar disorder.

Keywords: stroke, emotional incontinence, bipolar disorder

Introduction

Emotional incontinence (EI) is an increase in the frequency of crying or laughing episodes.¹ On the other hand, post-stroke mania is a rare neuropsychiatric complication that is observed in less than 1% of stroke cases.² Depression is the most frequently occurring psychiatric disorder after stroke, and secondary bipolar disorder is considered an uncommon consequence and it is only reported anecdotally.² Post-stroke EI and bipolar disorder are two disorders that involve the dysfunction of brain structures responsible for emotional regulation.³

We present a patient who developed post-stroke EI with multiple acute lacunar infarcts of lenticulocapsular and right semiovale regions. The patient experienced, after 6 months of antidepressant therapy, his first episode of hypomania, which is clinically managed by sodium valproate and low-dose paroxetine.

The objective of this work is to study the links between these disorders through a clinical case.

Case report

A right-handed, 43-year-old Tunisian man, without previous psychiatric history, presented with frequent and easily provoked spells of emotions (typically manifested by inappropriate laughing and crying). The patient was a nonsmoker and a teacher.

The patient had been crying for no reason more than ten times a day, but it was also observed that occasionally he could not stop laughing. According to his family, these brief fits of crying and laughing occurred in situations appropriate to the context and appeared like an uncontrolled exaggeration of a normal mood. These symptoms

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appeared after an ischemic stroke, 8 years ago. During the first year post-stroke, these symptoms were concomitant with partial somatic seizures (headache) diagnosed by a neurologist. These seizures were treated with carbamazepine. This treatment was stopped after 5 years of evolution without seizures. However, the gradual worsening of emotional disorders and their effect upon social and professional life led his family to make an appointment in our outpatient psychiatry service.

During the mental examination at admission, the patient presented with good appearance, was alert, cooperative, oriented in time and space, and had passive dependent personality traits. There were no manic and depression symptoms. Neurological examination revealed discrete hemiparesis in the left hemibody. The Mini-Mental State Examination score was 28/30.⁴

Brain magnetic resonance imaging (MRI) showed the presence of multiple acute lacunar infarcts of lenticulocapsular and right semiovale regions. Electroencephalogram was normal during excessive laughing and crying.

Authors ran a brief test battery to exclude other organic causes, including blood chemistry, thyroid function, folic acid, vitamin B12, CRP, summary analysis of urine type II, coagulation studies, illicit drugs in urine and alcohol in blood, chest radiography, HIV, hepatitis C and hepatitis B viral markers, and syphilis serology, which revealed no significant alterations. EI was diagnosed.

Treatment with sertraline (50 mg) was initiated. The EI symptoms improved remarkably. After 6 months he presented with hypomanic symptoms such as expansive mood, decreased need for sleep, increased goal-directed activity. MRI did not reveal another stroke. So, we made adjustments in pharmacotherapy to valproic acid (1,000 mg/day) and benzodiazepine. He remained euthymic in the next 5 months of follow-up until he progressively presented with worsening of EI. We proceeded with another pharmacotherapy adjustment adding paroxetine 10 mg once a day. Four months later, he was asymptomatic. Medication with low-dose paroxetine was continued. Written, informed consent was obtained from the patient.

Discussion

The classic literature on pathological laughter and crying emphasizes the difference between post-stroke EI and lability of affect in post-stroke mania or in bipolar disorder.³ Manic symptoms include expansive and/or irritable mood, decreased need for sleep, increased goal-directed activity, recklessness, disregard for social constraints, talkativeness, racing thoughts, excessive laughter or giggling, and poor judgment. In a large

community study that investigated the prevalence of neuropsychiatric disorders after stroke, secondary mania or bipolar disorder was not observed.² So, as a consequence of the paucity of post-stroke mania, it is difficult to determine precisely the clinical, demographic, and prognostic features.²

The prevalence of EI has been reported to be 15%–34%.^{1,5} EI is characterized by brief, but frequent and intense episodes of uncontrollable crying and/or laughing. The relationship with environmental stimuli is unclear, but these symptoms can be triggered by nonspecific stimuli.¹

It has been shown that post-stroke EI is related predominantly to serotonergic system dysfunction.⁵ The efficacy of antidepressant treatment for EI is well-established, and selective serotonin reuptake inhibitors (SSRIs) are the first-line drugs of choice.³ Many post-stroke EI cases showed a rapid response to SSRI treatment, as in our case.

It is interesting to note in the present case, the improvement of hypomanic episodes with mood stabilizer, and EI with low-dose SSRIs. For the treatment of this condition, some drugs including olanzapine, lithium, carbamazepine, and acid valproic have been tried with variable or no response.⁵ In our case, a successful outcome was achieved with valproic acid and low-dose SSRIs. These results suggest that the course of post-stroke EI and prognosis for successful treatment after brain injury can be different in subjects with EI or with a combination of EI and bipolar disorder. The appearance of a pharmacologically induced hypomania in this patient could suggest a link between EI and bipolar disorder. In this case, MRI showed right-sided stroke and the presence of multiple acute lacunar infarcts of lenticulocapsular and right semiovale regions. Kim⁶ mentions that among the lesions involving mainly the globus pallidus, dorsally located lesions are more often associated with EI than ventrally located ones. Many studies^{1,6–8} conclude that lenticulocapsular stroke of the basal ganglia/internal capsule in the right anterior region and the pons which contain abundant serotonergic fibers were closely related to EI. But, other studies suggest that the frontal lobe (including the orbitofrontal cortex), temporal lobe, basal ganglia, thalamus, and right-sided stroke are involved in post-stroke mania.^{2,9,10} Although the majority of the studies reporting secondary mania after stroke allocate lesions to the right hemisphere,^{11,12} a putative effect for laterality on its etiopathogenesis remains under discussion.^{9,13} A hypothesis for the development of this condition is that dysfunction of the fronto-limbic circuits negatively influences mood modulation, eventually resulting in manic attacks.^{14,15} Preexisting subcortical atrophy and a family history of bipolar disorder are also related to post-stroke mania.^{14,15}

These observations suggest that, at least during the acute stage of post-stroke, EI is more closely related to neurochemical changes associated with damage to specific brain regions. The brains of patients with post-stroke pathological crying had low baseline serotonin-binding potential.¹⁶ Also, metabolic hyperactivity in the basal ganglia in bipolar disorder is reported in the literature.² Many studies^{17,18} have examined the neural substrates underlying the manic state, and have found evidence of decreased right rostral and orbital prefrontal cortex activation. In addition, there is an increased activity in left dorsal anterior cingulate and the left head of caudate. This part is also associated with pathological laughter and crying after neurological lesions.

The existence of a right-sided stroke could have favored the occurrence of hypomania in the case of our patient. This illustrates that EI and bipolar disorder sometimes involve structures of the emotion circuit.

Limitations

Unfortunately, we could not perform any functional imaging like single photon emission tomography, as we do not have it in our hospital.

Conclusion

EI, post-stroke mania, and bipolar disorder sometimes involve structures of the emotion circuit. More caution regarding antidepressant treatment is required because of the potential risk of pharmacological hypomania, especially if there is a right-sided brain lesion. The clinician should carefully explore any history of bipolar disorder and/or personality disorder characterized by mood instability.

Disclosure

An abstract for this paper has been previously published in Mnif L, Masmoudi J, Charfi N, Baâti I, Kolsi S, Jaoua A. Emotional incontinence or bipolar disorder? About a case report. *European Psychiatry*. 2011;26(Supplement 1, Abstracts of the 19th European Congress of Psychiatry):230. The authors have no conflicts of interest to disclose.

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