Late Onset Mania after the Removal of Right Anterior Temporal Lobe Oligodendroglioma

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Abstract

The pathogenesis of bipolar disorder is multi-factorial and not fully understood, having polygenetic inheritance which may cause changes in neurotransmitter systems and dysfunction of fronto-limbic neuronal circuits. We aim to present a case which supports the hypothesis that right hemispheric hypofunctioning may contribute to the pathogenesis of bipolar disorder. Our manuscript includes a case of a 62-year-old male with new onset mania following resection of right sided temporal oligodendroglioma, suggesting that the hypofunctioning of this hemisphere may have lead to increased blood flow and activation of the left hemisphere.

Case Presentation

A 62-year-old, right-handed male was admitted following new onset confusion and mood disturbances. He had no prior history of mood disorder. While hospitalized, he experienced seizures, and a brain MRI revealed a right temporal lobe mass. The patient underwent right anterior temporal lobe resection and subsequent radiation therapy for an oligodendroglioma. (See Figure 1)

Following surgery, he complained of mild memory deficits. Mini Mental status exam was 28/30. He had right sided facial numbness but no other focal neurologic deficits. He was treated with levetiracetam and had no recurrence of seizures. Despite good neurologic outcome, the patient continued to have significant change in his mood and behaviors. He experienced periods of elevated mood with insomnia, increased goal directed activity and irritability. These episodes would last for periods of several weeks. He reported paranoid thoughts about possible governmental conspiracies and had grandiose delusions that he was the only person who could intervene. On several occasions, he placed himself in potentially dangerous situations and demonstrated a lack of fear for his own safety.

The patient lacked insight into the inappropriateness of his behaviors. He did not experience depressive symptoms, anxiety, hallucinations, suicidal or homicidal thoughts.

Five years after surgery he was involuntarily admitted for manic symptoms, after unexpectedly driving “about 3000 miles” in five different states for no apparent logical reason. On exam, he was cheerful with rapid and loud speech, increased sense of self-worth and circumstantial thought process. Levetiracetam was discontinued and valproic acid was started. Quetiapine and alprazolam were used to help improve sleep and paranoid thoughts. During the next two years, the patient continued...
to display a cheerful affect with increased sense of self-worth, but required no further hospitalization.

Discussion

The mean age of onset for bipolar disorder (type I and type II) ranges from 17-31 years of age, with new onset disease becoming increasingly rare beyond the age of 40. In the elderly, new onset mania has been documented to be caused by organic disease in up to 43% of cases. For this reason, new onset mania in elderly patients should lead clinicians to consider further testing including neuroimaging and EEG to rule out organic causes. Stroke, CNS infections, frontotemporal dementia, drug use and thyroid disease have been implicated in secondary mania. The mechanism by which mania develops in the presence of a focal cortical lesion is unknown and likely multi-factorial. However, damage to either the right frontal-temporal circuit or the right frontal-subcortical circuit has been implicated in many case reports. Indeed, perfusion studies suggest that secondary mania can be caused by "contralateral release phenomenon" with increased activation of the left hemisphere in the setting of right hemispheric damage. Patients with primary bipolar disorder have also been shown to have relative increase in activity of the left frontal lobe on EEG and fMRI during manic episodes. In contrast, patients with unipolar depression are characterized by a relative decrease in left frontal EEG activity and decrease in left prefrontal cortical metabolism. Although most patients with secondary mania show right hemisphere lesions, there are patients who develop mania secondary to left sided lesions which may reflect individual differences in patterns of functional lateralization.

Functional MRI studies have demonstrated differences in executive functioning when comparing the right vs left prefrontal cortices. Regions within the right frontal cortex are more involved with inhibition of inappropriate motor impulses and responses. These activities involve circuits connecting the right prefrontal cortex with the right anterior temporal lobe and amygdala. These circuits are involved in threat detection and harm avoidance. In contrast, regions within the left prefrontal cortex show increased activation during approach behavior and reward seeking. Functional MRI studies and case reports of secondary mania support the theory that mania is caused by dysregulation of prefrontal cortical circuits with an excessive approach-related affect (reward seeking) and decrease in behavioral inhibition (harm avoidance) due to relative increase in left prefrontal cortical activity and decrease in right PFC activity.

In this case report, the patient developed recurrent manic symptoms following the resection of a right anterior temporal lobe oligodendroglioma. He had not experienced any mood symptoms prior to surgery. Following surgery, the patient had no major neurologic symptoms and no recurrence of seizures. He responded well to valproic acid and quetiapine without recurrence of manic symptoms during a two-year follow-up period. The location of the patient’s tumor and subsequent resection of the right anterior temporal pole and right amygdala would have caused a significant disruption of the right frontal-limbic circuit. This case contributes to other case reports which demonstrate postoperative mania following right temporal lobectomy.

Conclusion

Our case demonstrates the possible role of cortical imbalance in primary forms of bipolar disorder and serves as a reminder that secondary causes of mania should be considered before diagnosing the elderly with primary bipolar disorder.

References
