The Cranial Rhythmic Impulse as a Measure in Patients With Bipolar Disorder: A Case Report

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CASE REPORT

Abstract

Bipolar disorder is a severe psychiatric illness associated with profound impairment in social and occupational functioning. Several conditions, including psychiatric disorders, have been associated with a decreased cranial rhythmic impulse (CRI). In this case report, a 20-year-old man with a recent manic episode is found to have a normal cranial rhythmic impulse, which is found to be decreased following resolution of the manic episode. This case illustrates the potential that a relative CRI increase in bipolar disorder may be a sign of an active manic state.

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Introduction

The prevalence of bipolar disorder in the United States is 0.6%, with a male-to-female ratio of 1:1.1 and a mean age of onset of 18 years old.[1] Pharmacologic treatments and psychosocial interventions are the mainstay.[2] There is a paucity of articles that discuss assessment and possible treatment options for psychiatric illnesses such as bipolar disorder with osteopathic manipulative treatment (OMT).[3] The cranial rhythmic impulse (CRI) is reported to be decreased in acute psychiatric conditions compared to the normal CRI within the populace.[3] The current case report describes a young man with bipolar disorder who is found to have a notable relative increase in his cranial rhythmic impulse during an acute manic state compared to his euthymic state.

Case Report

A 20-year-old man was hospitalized for acute euphoric mood that lasted 7 days, associated with impulsive spending, grandiosity, racing thoughts, increased activities, decreased need for sleep, and increased talkativeness with paranoid delusions. He had met criteria for bipolar disorder 1, severe with psychotic features.[1] Laboratory and diagnostic imaging studies completed during psychiatric hospitalization were unremarkable and included a urinalysis, complete metabolic panel, complete blood count, thyroid stimulating hormone level, rapid plasma reagin, folate level, Vitamin B12 level, drug screen and a CT of the head.

Upon discharge and initial evaluation in the clinic, the patient denied any other episodes of hypomania or mania and no significant episodes of depressive symptoms. He had no notable medical or family history. Social history was significant for marijuana use; last use was 3 months prior to his manic episode, and no other tobacco, alcohol, or illicit substance use was reported. Medications at discharge included ziprasidone, 40 mg twice a day; divalproex ER, 500 mg twice a day; and benztropine, 1 mg twice a day.

Physical examination

Osteopathic structural examination findings included a CRI of 12 cycles per minute; C6 ERLSL; T6 FRRSR; L1-3 SRRL; positive left standing flexion test; and left posteriorly rotated innominate.

Mental status examination was notable for the patient being alert and oriented, no mannerisms or motor hyperactivity, mood reported as "rested," a full affect range, speech non-pressured, no suicidal or homicidal ideation, no hallucinations or delusions, as well as fair insight and judgment.

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Treatment

The patient was continued on his current medication regimen, and OMT was used to address somatic dysfunction. At 1 week follow-up, the subject presented with continued mood stability, denying any manic symptoms, but he reported severe daytime somnolence from the medications. To address the reported side effect, ziprasidone was reduced to 40 mg at bedtime.

Within 4 days the patient experienced a relapse manic episode and was psychiatrically hospitalized again. Ziprasidone was replaced with risperidone 2 mg twice a day while the divalproex and benztropine were continued. Upon follow-up, the patient divulged that, on further reflection, he'd concluded his mania was not controlled on his initial visit, nor at the time of his ziprasidone dose reduction. His repeat osteopathic structural examination was notable for a CRI of 8 cycles per minute.

Discussion

The normal CRI biphasic cycle of motion is reported to have a rate of 10 to 14 cycles per minute.[4,5] Internal and external factors have been found to alter the CRI.[3,4,5] Individuals receiving first generation antipsychotic medications were noted to have no significant changes in CRI.[6] However, anecdotally, many osteopathic physicians, including this author, have encountered a reduced CRI in patients who are taking psychopharmacologic medications, particularly antidepressants, mood stabilizers, and antipsychotics. To date, to the best of this author's knowledge, no one has reported a relative increase in CRI in association with a manic state in an individual with a psychiatric illness such as bipolar disorder.

An increase in the CRI has been associated with vigorous physical exercise, fevers, and cranial OMT. A decrease in the CRI has been reported with many disease states including physical and psychological stress, chronic fatigue, infections, depression and other psychiatric conditions, and chronic poisoning.[3,4,5] In addition, the rate and amplitude of the primary respiratory mechanism (PRM), as manifested by the CRI, is a diagnostic and prognostic indicator of body unit compromise and response to treatment. A decreased rate and low amplitude suggest a compromised body unit.[5]

The PRM is manifested palpably as the CRI and was first described by the clinical team of John Woods, DO, and Rachel Woods, DO.[3] Neuroimaging findings in patients with bipolar disorder have included abnormalities of prefrontal-striatal-thalamic circuits, amygdala and midline cerebellum. Functional imaging findings include increased activity of the anterior cingulate gyrus, striatum, thalamus, and amygdala.[7]

First-line pharmacologic treatment for those with an acute manic episode includes mood stabilizers (such as lithium, valproic, acid or carbamazepine) or second-generation antipsychotics (such as olanzapine, quetiapine, and ziprasidone) and often psychiatric hospitalization. Medication doses can be rapidly titrated on an inpatient setting and polypharmacy commonly occurs.[2] Despite medication compliance, it can be hard for those with bipolar disorder to recognize they are in a manic state.

The patient here has been dependent on family and friends for identification of manic states. During his initial visit, this safeguard was not successful as the patient reports in hindsight he was downplaying his mania. His physical exam at that time did not reveal a decreased CRI. Fewer factors are associated with an increased CRI.[7] There have been no literature reports of any psychiatric condition being associated with a normal or increased CRI.

This case report found a normal CRI in a patient reporting and presumed recovered from an acute manic episode, later further exacerbated by lowering medication. An active manic episode can fully manifest once a medication dose is lowered. This relapse is at increased risk if the initial manic episode has not fully resolved.[8]

Osteopathic medicine approaches each patient through the osteopathic philosophy.[9] Osteopathic palpatory skills may have a role in assessing psychiatric patients with bipolar disorder. Whether a normal CRI found in a bipolar subject in acute recovery from a manic episode can help to discern and guide management decisions may need to be further explored.

Conclusion

This case illustrates the potential that a relative CRI increase in bipolar disorder may be a sign of an active manic state. Osteopathic palpation of the CRI may provide an additional measure to monitor and guide treatment in acute manic episodes. Additional research is needed to determine baseline CRI in specific psychiatric disorders, acute changes during subtherapeutic states and the use of CRI as a potential method of monitoring disease course.

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