Case report

eISSN 2799-8010
J Yeungnam Med Sci 2022;39(1):58-61
https://doi.org/10.12701/yujm.2020.00864

JYMS
JOURNAL OF YEUNGNAM MEDICAL SCIENCE

Mega cisterna magna in bipolar mood disorder: a case report

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Mega cisterna magna (MCM), one of the members of the Dandy-Walker complex, is a developmental malformation of the posterior fossa that is larger than 10 mm but morphologically does not affect the vermis and cerebellar hemispheres. Reports of psychiatric disorders associated with this anomaly are rare. We present the case of a patient with MCM who presented with a psychotic manic attack and was diagnosed with bipolar disorder. A 28-year-old female, single housewife, university graduate, presented with irritability, decreased sleep and appetite, distraction, and agitation. The patient also had a delusion of reference. In the clinical follow-up, an increase in energy and an increase in the amount of speech were observed. Her neurological examination was normal, and cranial magnetic resonance imaging revealed an MCM. The relationship and clinical significance of MCM with psychosis and mood disorders have not yet been fully elucidated. It is not known whether this association is accidental or based on etiological commonality. The purpose of this case report is to review the relationship between the cerebellum and psychiatric symptoms and to contribute to the literature.

Keywords: Cerebellum; Mania; Mood disorders; Posterior cranial fossa; Psychotic disorders

Introduction

Bipolar disorder is a chronic disease with elevation, depression, and recovery periods and is accompanied by psychotic symptoms during episodes of illness. The cerebellum is thought to play a role in the pathophysiology of bipolar disorder. It is located in the posterior cranial fossa, behind the fourth ventricle, pons, and medulla oblongata. The tentorium, which is an extension of the dura mater, separates the cerebellum from the cerebrum. The cerebellum, which is the largest structure of the central nervous system after the cerebrum, has anatomically and physiologically different functional parts and consists of highly ordered neuronal units sharing the same basic cerebellar microcircuit [1]. The cerebellum is responsible for coordinating movement, maintaining balance and posture, muscle tone, and motor learning. In addition, the cerebellum is linked to many brain areas related to cognition and behavior, such as the dorsolateral prefrontal cortex, medial frontal cortex, anterior cingulate, and posterior hypothalamus, particularly through the thalamus. It is thought that noradrenergic, serotonergic, and dopaminergic afferents from the nuclei in the brainstem may play a role in the regulation of sensory, procedural, linguistic, and emotional activities through cerebellar connections with limbic and cortical association areas. This information suggests that the cerebellum contributes significantly to mood regulation and that cerebellar

Received: December 8, 2020 • **Revised:** March 5, 2021 • **Accepted:** March 6, 2021
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anomalies may be involved in the pathophysiology of mood disorders [2].

Cisterna magna is the subarachnoid space located behind the medulla oblongata under the cerebellum. This space allows the cerebrospinal fluid (CSF) to be transferred from the fourth ventricle to the foramina. Mega cisterna magna (MCM), which is a developmental malformation of the posterior fossa, is known for the cisterna magna being larger than 10 mm, morphologically normal vermis and cerebellar hemispheres, and no hydrocephalus [3,4].

MCM is observed in approximately 1% of postnatal brain images. The incidence of isolated MCM in the community is not clearly known, as it is asymptomatic. The coexistence of MCM and psychiatric disorders is rare and possibly coincidental. However, the relationship between these two is striking when evaluated in terms of the effect of the cerebellum on psychiatric symptoms and mood regulation, and more studies are needed to reveal the causal relationship between them.

This study aimed to present the case of a patient who presented with a manic episode of bipolar disorder associated with MCM, to review the relationship between the cerebellum and psychiatric symptoms, and to contribute to the literature.

Case

Ethical statements: Ethical approval for the study was obtained from the non-invasive ethics committee of Sakarya University Faculty of Medicine (No: 02.03.2021-E.14816).

A 28-year-old female, single housewife, university graduate, was brought with complaints of irritability, decreased sleep and appetite, distraction, reference delusion, and agitation that started 10 days before her hospitalization. The patient, who was referred to our service with a prediagnosis of psychotic attack by a psychiatry consultant doctor in the emergency department, with a haloperidol injection, had reduced self-care, looked agitated, and was sleepy. The patient, who did not have a psychiatric history before, had a hypomania attack 2 years ago for 2 to 3 days with complaints of irritability, decreased sleep and appetite, decreased attention, and concentration.

On mental state examination, the patient was conscious, oriented to place-time-person, and was not willing to communicate, but could establish eye contact. The agitated patient's affect was labile, and her mood was irritable. Her speech was reduced and fit for purpose, and her tone of voice was natural. She spoke defensively. The flow of thought and associations was accelerated. Her attention and concentration were reduced. The patient's thought content had a reference delusion that people were texting her on television. Her perception was not disordered. Near and distant memories were normal. Knowledge and intelligence were correlated with the education level. Abstract thinking was natural. Judgment and insight were reduced. Her psychomotor activity was increased. Neurological deficits were not observed during her neurological examination. There was no history of smoking, alcoholism, or other substance abuse. No significant pathological findings associated with the perinatal history were observed. The patient was born through vaginal delivery at term. She had no known disease or hospitalization history during her infancy. Her psychomotor development was normal. No family history of any neurological or psychiatric illness was found. For psychological assessment, the Minnesota Multiphasic Personality Inventory (MMPI) and intelligence quotient (IQ) tests were performed on the patient. The IQ test results were in the normal intelligence range. In the MMPI personality test, a psychopathological increase was not observed in the subtests and the general test profile.

In the first week after her admission to our service, while her anamnesis was taken, the patient had almost no speech and insomnia. On the clinical impression of the patient, elevated mood, increased amount of speech and energy, distraction, thought flow, and acceleration in associations were observed. The patient was diagnosed with bipolar disorder manic episode.

The hemogram and biochemical values were within normal limits. Replacement therapy was initiated for low vitamin D levels. In cranial magnetic resonance imaging (MRI), a 2 cm wide MCM variation with the same intensity as the CSF in the T1A and T2A sequences was detected in the infravermian area, extending posteriorly between both cerebellar hemispheres (Fig. 1).

The patient was started on quetiapine (100 mg), haloperidol (10 mg), and biperiden (2.5 mg) injection therapy. On the third day of hospitalization, 10 mg of olanzapine was added to the treatment. The dose of olanzapine was gradually increased to 20 mg over 2 weeks. The doses of haloperidol and biperiden were tapered. During the third week of the hospitalization, the patient's psychotic symptoms regressed and 300 mg of lithium was added to the treatment for the ongoing mood symptoms. When the lithium dose was gradually increased to 900 mg, the patient's affective symptoms regressed, and the blood lithium level was measured as 0.72 at the time of discharge. The patient, who was in a good clinical condition and had no homicidal or suicidal thoughts, was discharged.

Discussion

According to the Diagnostic and Statistical Manual of Mental Dis-

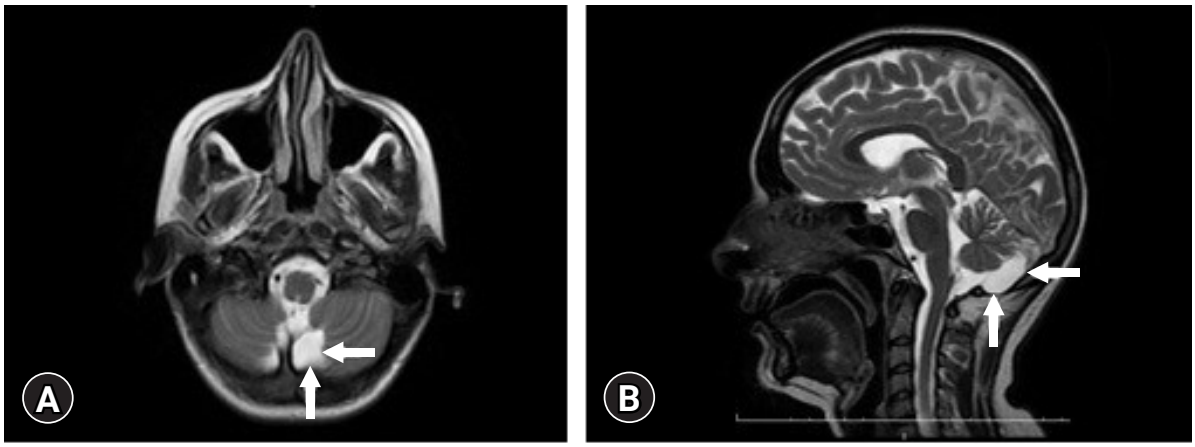

Fig. 1. The unenhanced T2-weighted brain magnetic resonance imaging scans of (A) the axial and (B) sagittal planes show mega cisterna magna (arrows) with the same intensity as the cerebrospinal fluid extending from the interhemispheric area to the posterior, which is more prominent in the left cerebellar hemisphere.

orders, fifth edition (DSM-5) criteria, affective episodes lasting longer than 1 week (shorter if resulted in hospitalization) are diagnosed as manic episodes of bipolar mood disorder [5]. This case meets the bipolar disorder criteria in terms of symptom severity and duration according to the DSM-5 criteria. The patient did not have any known alcohol or psychoactive substance use. This was confirmed by toxicological analysis of the urine. Moreover, no anomaly was found in the vermis and cerebellar hemispheres on MRI, and no neurological deficits were detected. These findings are consistent with the diagnosis of MCM. When a search was conducted using the keywords 'mega cisterna magna' on Google Scholar and PubMed databases, it was found that this anomaly is associated with psychiatric disorder; Langarica and Peralta [6], Ferentinos et al. [7], Turan et al. [8], Kumar et al. [9], Karayılan and Erol [4], Kani et al. [10], Erzin [11], Balcioğlu et al. [12], and Öztürrk et al. [13]. Only two of these case reports were associated with manic episodes; Turan et al. [8] and Öztürrk et al. [13]. Previous case reports have mentioned that there may be a relationship between MCM and psychiatric symptoms. This relationship is more evident in malformations affecting the vermis and cerebellar hemispheres [14,15]. However, in proton magnetic resonance spectroscopy, it was observed that the levels of gamma-aminobutyric acid (GABA) were decreased in the cerebellar tissue of patients diagnosed with unipolar and bipolar disorder. Another postmortem study showed that Purkinje cells were significantly reduced in the frontal lobe of the cerebellum in a patient with bipolar disorder. It has also been suggested that abnormal GABA proteins play an important role in the expression and migration of GABAergic Purkinje cells during cerebellar development [16-18].

Although there is no cerebellar parenchymal anomaly that can be detected by imaging and neurological examination, postmortem findings of MCM as a developmental anomaly have been investigated in cases such as stillbirth, but there is no postmortem neuropathological study of adult patients with MCM [19]. Although this case cannot be evidence of a causal relationship alone, the relationship between the neuroanatomical location of the variation and mood regulation and psychiatric symptoms brings to mind an underlying etiology that may point out a causal relationship. Therefore, further studies are needed to establish a causal relationship between the effects of structural disorders, such as MCM, on cerebellar mechanisms and psychiatric disorders. These studies may be particularly valuable in guiding studies on the pathophysiology and treatment of mood and psychotic disorders.

Notes

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

Author contributions

Conceptualization: EY, YG, EMK, ABY; Investigation: ABY; Data curation: SK, YG; Formal analysis: YG, ABY; Methodology: EY; Supervision: YG, ABY; Writing - original draft: EY; Writing - review & editing: all authors.

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