Comorbid schizophrenia and obsessive compulsive disorder associated with mega cisterna magna: a case report

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INTRODUCTION

Historically, the cerebellum has been regarded as the region of the brain responsible for motor coordination, balance, gait, and fine motor control. Today, novel findings point to the importance of the cerebellum’s involvement in the pathophysiology of psychiatric disorders.¹ There is converging evidence suggesting that a cerebellar dysfunction could underlie some of the psychiatric and neurological symptoms as well as cognitive dysfunctions observed in schizophrenia.² The involvement of cerebellar dysfunction has also been suggested in the pathogenesis of Obsessive–Compulsive Disorder (OCD) and schizophrenia-OCD comorbidity.³ Dandy-Walker complex (DWC) is a series of anomalies in the posterior fossa, including Dandy–Walker malformation, Dandy–Walker variant, mega-cisterna magna and posterior fossa arachnoid cyst. Mega cisterna magna is the mildest form of “Dandy-Walker complex” and a developmental variation of the posterior fossa characterized by the enlargement of the cisterna magna, morphologically intact vermis and cerebellar hemispheres.⁴ To the best of our knowledge, there is only one case report in the literature describing a patient with the Dandy Walker complex and schizophrenia comorbid with OCD. Our aim here is to present a case of schizophrenia comorbid with OCD and mega cisterna magna, successfully treated with clonazepam.

CASE REPORT

The patient, a 57 year-old woman, attended to our psychotic disorders outpatient clinic because of her fear of talking involuntarily and being overheard by other people. She mentioned that she had an urge to utter some annoying sentences such as “the cars should crash into each other, my father should die”, which she had difficulty in controlling and which led to a sense of guilt. To prevent this condition she was repeating some meaningless words in her mind, which could last for several hours per day. In her history there were several hospitalizations with psychotic symptoms including somatic, paranoid and persecutory delusions over a period of 30 years. There was no history of psychiatric disorders in the patient’s family. No alcohol or substance misuse was reported. Her mental examination revealed doubt and aggression obsessions accompanied by mental compulsions and visual hallucinations. Her affect was blunted and she exhibited partial insight into the validity of her beliefs. In her neurological examination there was no significant finding and electroencephalography was unremarkable. Cranial Magnetic Resonance Imaging (MRI) scan disclosed “mega cisterna magna” in the midline of the posterior occipital region (2.6×3.1 cm) (Figure 1). She had been taking 4 mg risperidone and 300 mg quetiapine per day for several years. She was diagnosed with both schizophrenia and OCD. She had 53 points in PANSS and 21 points in YBOCS. According to the neuropsychological function tests the full scale IQ was 80. Neuropsychological assessment showed moderate deficit in selective attention, mild deficits in visual and working memory, and impaired abstract reasoning and judgement. Clomipramine was started and increased to 225 mg/day over seven months. With no significant improvement at the end of seven months clomipramine was changed to fluvoxamine and increased to 300 mg/day but despite adequate duration again no difference was observed in the patient. After this regimen, 1 mg/day clonazepam was added to fluvoxamine and a significant clinical improvement with a decrease of YBOCS to 1 was recorded after five days.

DISCUSSION

It is well established that OCD is a common disorder in patients with schizophrenia with a 13.6% prevalence.⁵ Although the exact etiology remains poorly understood, neuroimaging studies imply that a particular neuroanatomic and neurobiological process may be involved
in the comorbid group. In literature there is evidence that cerebellar dysfunction may play a role in the cognitive symptoms which are observed in patients with schizophrenia. Also, cerebellar abnormalities such as gray matter volume changes have been reported in patients with OCD. To our knowledge, while there are several case reports of psychotic disorders, only one case of comorbid OCD-schizophrenia associated with mega cisterna magna has been reported. In our patient, similar to the previous report, there were disruptions in attention, abstract reasoning and judgment but there was no deterioration in memory or mental arithmetic. In our patient, a significant improvement was observed in obsessive compulsive symptoms with the administration of clonazepam with no recurrence after a three month follow up.

GABAergic abnormalities are involved in the development of schizophrenia especially in the cognitive disruptions of the disease. Also, GABA neurotransmission disorders have been noted through the cortical inhibitory processes in OCD. It is well known that clonazepam increases GABAergic function and inhibitory processes by GABAergic neurons, which are largely located in the cerebellum and neocortex. In this case, mega cisterna magna, schizophrenia and OCD symptoms may occur coincidentally or any cerebellar dysfunction due to mega cisterna magna may contribute to the occurrence of some psychotic, cognitive symptoms and obsessions. Such neurostructural variants may offer an insight into a better understanding of the neurodevelopmental models underlying schizophrenia comorbid with OCD, and clonazepam may be an effective treatment option in resolving treatment-resistant obsessive compulsive features in schizophrenia.

REFERENCES


