

ARNOLD-CHIARI MALFORMATION IN A PATIENT WITH BIPOLAR I AND PANIC DISORDERS

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Arnold-Chiari malformation (ACM) encompasses a group of deformities of the hindbrain characterized by displacement of one or two cerebellar tonsils by 5 mm (or greater) caudally to the foramen magnum. Four subtypes of ACM have been described. ACM-1 is characterized by a herniation of the cerebellar tonsils into the foramen magnum, sometimes associated with syringomyelia, hydrocephalus, or skull base alterations. Although comorbidity between cerebellum disorders and psychiatric syndromes has been described in patients with a main diagnosis of cerebellar disorders (Schmahmann et al. 2004), little is known about this condition in ACM-1 patients. We describe herein the case of a 36-year-old female patient suffering from Bipolar I (BP-I) and Panic Disorders (PD), who had been incidentally diagnosed with ACM-1. The patient provided an appropriate written consent both for clinical and research purposes. Moreover, the manuscript does not contain any identifiable patient information, ensuring and protecting patient's anonymity.

Ms A was a 36-year-old Caucasian, single woman, suffering from paucy-symptomatic panic attacks and seasonal mood dysregulations since her childhood. The first clinically relevant symptoms started abruptly when she was 28, concomitantly with work disputes. The patient experienced anhedonia comorbid with panic attacks, depersonalization, derealization, and persistent headache. Symptoms gradually increased, with dizziness, facial dysesthesia, photophobia and diffuse pain sensations. Ms A referred to a psychiatrist who diagnosed a major depressive episode (MDE) comorbid with Panic Disorder (PD), and prescribed escitalopram (20 mg/day) and prazepam (10 mg/day), with a partial response. When she was 35 (again concomitantly with work disputes), she suffered from headache, dysphoria, panic attacks, restlessness, and severe paranoid ideation evolving to persecutory delusion. She was treated with aripiprazole and pregabalin (at unspecified dosages) both ineffective. Subsequently, she was treated with lithium, vortioxetine and olanzapine (at unspecified dosages), but symptoms worsened towards severe sleeplessness (with sleep apnea syndrome), and auditory hallucinations, so that she was admitted in our inpatient section. She was diagnosed with BP-I disorder (current mixed episode with psychotic symptoms) and comorbid PD. She complained about auditory complex misperceptions, and persistent facial dysesthesia, with photo-

phobia. A brain MRI was performed, while considering the persistence of somatic manifestations and the overall treatment-resistance. MRI revealed a congenital ACM-1, with a low commitment of the cerebellar tonsils in the foramen magnum (> 3 mm), with no indication for decompression, according to neurosurgical evaluation. She was treated with valpromide (900 mg/die), pregabalin (450 mg/day), and risperidone (6 mg/day), with a significant improvement of insomnia, but with no response of both psychotic and somatic symptoms. She was then discharged and followed-up in the outpatient section. After three months, she showed an improvement in the mood symptoms, with no change of psychotic somatic complaints.

As far as we know, five case reports of ACM-1 in psychiatric patients have been already described: two in anxiety disorder patients (Chisholm et al. 1993, Caykoylu et al. 2008), one in a patient with schizophrenia-like disorder (Iranković et al. 2006), and two in BD patients (Bakim et al. 2010, Hong et al. 2019). The case report published by Hong et al. (2019) was similar to ours, except for the relevance of somatic symptoms we found. It is difficult to ascertain if part of the somatic complaints of Ms. A were due to ACM-1 or to comorbid PD. However, it is worth noting that somatic symptoms were correlated to a sleep apnea syndrome, concordantly with previous findings in ACM patients (Dauvilliers et al. 2007). We are aware that, in our case, it is not possible to link univocally a clinical manifestation to one of the two conditions, except in a speculative manner. However, we believe that our case raises questions on how to deal with a rare clinical condition, hardly detectable and characterized by extreme variability in clinical presentation.

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