



sessions per day performed 1 hour apart, for 5 consecutive days. A 2mA current was used, with the anode placed on the left temporal-parietal junction and the cathode on the left dorsolateral pre-frontal cortex. The choice for this setup was based on a systematic review showing this as the most usual montage in schizophrenia, because it covers areas associated with both positive and negative symptoms. In addition, the precise places being stimulated can be mapped through computer modeling analysis or a neuronavigation system.⁵

After signing an informed consent form, the patient was started on the protocol. Clinical evaluations were performed on eight occasions using the Brief Psychiatric Rating Scale-Anchored (BPRS-A) and the Global Assessment of Functioning (GAF) scales: before the stimulation protocol, on the last day of the protocol and at months 1, 3, 6, 9, 12, and 15 after the end of stimulation. Also, medications remained stable along the 15 follow-up months. Figure 1 shows a robust reduction in overall BPRS-A and GAF scores during the 15 month-follow-up, during which no new tDCS sessions were applied. The BPRS-A score assessing auditory hallucinations had the highest reduction (ranging from 5 to 1). In the 9th month, the patient resumed work and was able to be alone in public places.

The synergistic effect between tDCS and psychotropic drugs has been recognized. According to the neuroplasticity hypothesis of tDCS, this technique may induce the release of neurotransmitters that increase the sensitivity of postsynaptic receptors, inducing subsequent cortical reorganization over time. This sustained effect over a prolonged period is more frequent in patients with short duration of disease, higher educational level, and absence of substance abuse, as seen in this case report. It is postulated that the associated mechanism of action involves the correction of inhibition deficit mediated by GABA receptors.⁶

Despite the limited level of evidence, the present case does support the notion of a direct, positive tDCS impact, as shown by 1) rapid response, which had not yet occurred with use of antipsychotics only; 2) clinical response measured by BPRS-A, suggesting an impact of tDCS over time (Figure 1). Given the characteristics of this case, this approach requires further investigation before it can be used in different mental disorders. For that, prospective studies, different tDCS protocols and adequate follow-up evaluations must be designed.

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Disclosure

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First psychotic episode in an adult with Becker muscular dystrophy

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In the present report, I describe the case of a 50-year-old male diagnosed with Becker muscular dystrophy (BMD) six years earlier, prior heart transplant and progressive motor impairment, treated with everolimus and mycophenolate mofetil, and no previous psychiatric history. The patient was admitted to a psychiatric emergency room with persecutory and prejudice delusions, as well as ideas of reference; these symptoms had worsened over the past months. Secondly, he developed depressive mood, initial and intermediate insomnia, anorexia, passive death wishes, and decreased cognitive performance. He denied alcohol or recreational drug consumption. Both his brother and cousin had BMD and similar psychiatric symptoms.

No relevant changes had been detected on previous brain imaging. Laboratory and electrocardiographic evaluation

assessment at admission again did not show changes. Pharmacological treatment with aripiprazole 10 mg, mirtazapine 30 mg and alprazolam 0.5 mg was started. A month later, a new evaluation showed significant improvement of psychotic symptoms.

To the best of my knowledge, this is the first report of new onset psychotic symptoms in a middle-aged adult with BMD. A previous report has described a 23-year-old male with BMD and psychosis,¹ however the early onset suggests a comorbid primary psychotic disorder. In the present case, the late onset of symptoms indicates a secondary condition resulting from BMD progression. Thus, the case highlights a possible common etiological link between BMD and psychiatric disorders.

BMD is an X-linked recessive inherited disorder caused by mutations in the dystrophin gene located at Xp21, resulting in the production of abnormal but functional dystrophin.^{2,3} This cytosolic protein stabilizes the plasma membrane of striated muscle cells in skeletal and cardiac muscle. Mutation of dystrophin causes progressive muscle wasting and weakness, with onset usually in childhood.³


There is evidence that BMD might be associated with mental disorders and mental retardation,³ possibly because dystrophin is also expressed in the cerebral cortex as part of the neuronal postsynaptic apparatus.² The discovery of a dystrophin brain isoform provides an organic basis for the association of BMD with psychiatric disorders.³ Cosegregation of BMD and schizophrenia has been suggested – as proposed by Zatz et al.,⁴ this could either be the result of a gene contributing to the mental disorder, linked to the dystrophin locus, or else caused by an abnormality in the expression of the dystrophin gene, directly affecting the brain.

An increased prevalence of depression among BMD patients has also been reported,³ confirmed by a recent study adding attention deficit hyperactivity disorder, autism spectrum disorder, obsessive-compulsive disorder, and bipolar disorder to the mental health concerns of individuals with dystrophinopathies.⁵ Some studies have also described a relation between cognitive impairment and dystrophin deficiencies, given the lack of dystrophin isoforms Dp71 and Dp140 in the brain of affected individuals during development.⁵

Considering the evidence of an association between BMD and psychiatric disorders, as illustrated by the

present case, careful psychiatric and cognitive evaluation is essential in these patients, guiding diagnostic formulation and treatment planning and management. The literature on this subject is scarce, and thus further studies may contribute to a better characterization of psychopathology in individuals with BMD.

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