CLINICAL NOTE: Marchiafava-Bignami disease in a patient with schizophrenia and alcohol use disorder

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ABSTRACT

Introduction. Substance-related disorders are the most frequent comorbidity in schizophrenia. Concretely, alcohol is the most commonly consumed substance after tobacco. Patients with schizophrenia with this comorbidity have a worse clinical course and can develop serious neuropsychiatric complications. One of them, Marchiafava-Bignami disease (MBD) can be incorrectly diagnosed as a decompensation of their mental disorder.

Methods. A case of a 51-year-old man, diagnosed with schizophrenia and alcohol use disorder is presented. He experienced acute neuropsychiatric symptoms for which schizophrenia decompensation was suspected. Based on his atypical symptoms and medical history, a brain imaging test was performed and MBD was diagnosed.

Conclusions. MBD is an infrequent clinical entity that should be part of differential diagnosis in patients with alcohol abuse disorder experiencing atypical neuropsychiatric symptoms.

Keywords. Marchiafava-Bignami disease, schizophrenia, dual disorder, alcohol use disorders.

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CLASO CLÍNICO. Enfermedad de Marchiafava-Bignami en un paciente con esquizofrenia y trastorno por uso de alcohol

RESUMEN

Introducción. Los trastornos por abuso de sustancias son la comorbilidad más frecuente en la esquizofrenia, siendo el alcohol, concretamente, la sustancia más frecuentemente consumida tras el tabaco. Los pacientes con esquizofrenia que presentan esta comorbilidad presentan una peor evolución clínica y pueden desarrollar graves complicaciones neuropsiquiátricas. Una de ellas, la enfermedad de Marchiafava-Bignami (EMB), puede ser erróneamente diagnosticada como una descompensación del trastorno mental, conllevando graves consecuencias.

Método. Se presenta el caso de un varón de 51 años, diagnosticado de esquizofrenia y trastorno por consumo de alcohol. Experimentó síntomas neuropsiquiátricos agudos por los que se sospechó una descompensación de su esquizofrenia. Dada la presentación clínica atípica y sus antecedentes médicos, se sospechó una patología orgánica y se realizó una prueba de imagen cerebral en la que se diagnosticó una EMB.

Conclusiones. La EMB es una entidad clínica infrecuente que debe formar parte del diagnóstico diferencial en pacientes con trastorno por consumo de alcohol que experimenten síntomas neuropsiquiátricos atípicos.

Palabras clave. Enfermedad de Marchiafava-Bignami, esquizofrenia, trastorno dual, trastorno por consumo de alcohol.

INTRODUCTION

Substance-related disorders are one of the most frequent comorbidities of schizophrenia[1], being the alcohol the second most consumed substance after tobacco, with an use up to three times more prevalent than in the general population[2]. This comorbidity is associated with a worse prognosis, more hallucinatory and delusional symptoms, as well as a higher number of re-admissions, and rate of disability and suicide [1,2]. Furthermore, it is linked to the onset of other somatic diseases, some of which are infrequent but extremely severe, such as Marchiafava-Bignami disease (MBD)[3].

MBD is an encephalopathy characterized by a degeneration of the corpus callosum. It is mainly associated with chronic alcohol consumption and malnutrition[3]. Few cases have been described in the literature to date, being death the outcome in some of them[4]. Its clinical spectrum includes neurological and psychiatric symptoms (e.g., confusion, delirium, sensorial disturbances, decreased consciousness, memory impairment or disorientation) which appear in up to 80% of patients[5]. Clinical suspicion is essential, as it leads to its diagnosis through image techniques. Furthermore, its clinical features can be incorrectly interpreted as part of other neuropsychiatric diseases and, in patients with mental disorders, can lead the clinician to misdiagnosis[6,7]. However, to our knowledge, there are no data in the literature about the concurrence of MBD and other psychiatric disorders different to alcohol use disorder.

CASE REPORT

We present the case of a patient with schizophrenia and acute neuropsychiatric symptoms. We checked the clinical history in respect of his acute episode, the pre-morbid history and the subsequent clinical developments in a nine-month follow-up period. This case was discussed on the light of the existing literature.

A 51-year-old male, diagnosed with paranoid schizophrenia at approximately 25 years of age, was admitted to the hospital emergency department. In the last decade, his adherence to mental health care had been weak and he had maintained chronic and fluctuant positive symptoms of schizophrenia. He had been treated in the past with pipotiazine palmitate, and in the last six years, with fluphenazine decanoate and olanzapine. A change to paliperidone palmitate had previously been tried, but an intensification of his symptoms had led to a return to fluphenazine decanoate. However, in the previous three years before his admission, he had not received any antipsychotic treatment, until six months prior to the current episode, when he contacted Mental Health Services again and fluphenazine decanoate and olanzapine were reintroduced.

Regarding his social and functional situation, he had remained unemployed for more than a decade. He lived on his own, being independent for basic and instrumental activities of daily living.

Concerning his substance use history, he was a heavy tobacco smoker. Furthermore, he had been assessed by the Addiction Unit twelve years ago because of alcohol misuse, although no follow-up was carried out. Alcohol consumption worsened during the following years and acute intoxications were observed in some visits to his psychiatrist. At that time, hepatic steatosis was diagnosed and, in the last three years, he had presented several accidents related to alcohol consumption, which at that time was of 30 alcohol units per day, mainly consisting of wine. However, he had not received any specific treatment for alcohol dependence, except for a prescription of nalmefene that he did not take.

This patient was accompanied to the emergency ward by his father, who suspected a decompensation of his schizophrenia. He reported that the last dose of his depot antipsychotic had not been administered. The patient was assessed by the emergency medical team who, in line with this suspicion, consulted the psychiatry service. The relative, who had not seen his son for a month, informed that his speech had become incoherent and there was a severe worsening of his general condition, with weakness, anorexia and weight loss. Regarding the neuropsychiatric examination, there was a deterioration in his hygiene. He was partially oriented and had difficulties in maintaining attention and obeying simple orders, as well as being bradykinetic. His speech was sparing, incoherent and disorganised. Although it was not possible to exclude the presence of delusional thoughts and hallucinations, they were not main symptoms at that moment. Neurological examination revealed left hand mild action tremor and abolished Achilles reflexes, without other pathological findings. A blood test revealed 0.34 g/L of ethanol, hypertransaminasemia and a deficit of folic acid. A CT Scan revealed a homogeneous hypodensity at Corpus callosum (Figure 1), so admission to the Neurology department was decided.

During his admission, an MRI with intravenous contrast detected an osmotic demyelination, compatible with MBD (Figure 2). Treatment with vitamin and protein supplements was initiated and neurological symptoms improved slightly and he was discharge. In the months that have followed, the nutrient treatment and the antipsychotic treatment with fluphenazine decanoate, which was

Figure 1 Axial computed tomography of the patient showing a homogeneous hypodensity at Corpus callosum, compatible with Marchiafava-Bignami Disease

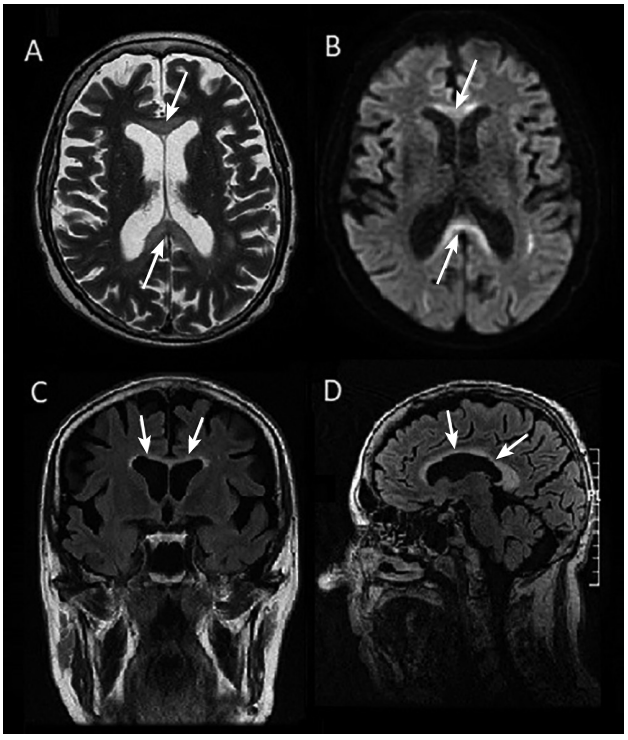

Figure 2 Magnetic resonance imaging of the patient showing osmotic demyelination signs in the corpus callosum area, compatible with Marchiafava-Bignami Disease. A) Axial Fast Spin Echo T2 (FSE T2); B) Axial diffusion-weighted imaging 1000b (DWI-100b); C) Coronal fluid-attenuated inversion recovery (FLAIR); D) Sagittal FLAIR.

not modified, has been continued. Despite alcohol intake has been reduced, abstinence has not been reached completely. Likewise, the autonomy and functionality of the patient is still moderately impaired and the necessity of a continued care has remained unchanged.

DISCUSSION

Patients with dual disorders generally suffer a worse clinical and social course of their illness. Concretely, in the case of schizophrenia, the frequent comorbid abuse of alcohol implies a potentiation of both conditions' adverse consequences[1,2]. The cognitive and functional impairment of schizophrenia is increased by the one caused by an acute or maintained alcohol consumption. Furthermore, this comorbidity causes worse social functioning and lower self-care, which can facilitate the onset of severe consequences such as Wernicke-Korsakoff disease or the infrequent entity called MBD[4].

These neurological conditions are characterized by symptoms than can be incorrectly interpreted as an acute psychotic decompensation[7,8]. In the case of EMB, although the presence of psychotic symptoms is rare[6], delusional and perceptive manifestations can be mistakenly oriented, with dramatic consequences[5]. Thus, a deep knowledge of these diseases is crucial for avoiding diagnostic errors. Specifically, the memory impairment, confusion and disorientation, as well as neurological and inflammatory signs, should raise suspicion over the existence of an organic disease and, then, image diagnosis tests should be carried out[9].

Moreover, this case exemplifies the importance of early intervention to avoid substance use and foster healthy nutritional habits in patients with schizophrenia, fostering specific plans for dual disorders which nowadays remain uncommon and heterogeneous[2].

CONCLUSION

In conclusion, in line with the existing literature, this case supports the importance of a comprehensive approach to patients with dual disorder. Specifically, EMB is a rare but serious clinical entity, that should be part of the differential diagnosis in patients with alcohol use disorder who experience atypical neuropsychiatric symptoms. This is particularly relevant in patients with other comorbid psychiatric disorder in which these symptoms can be mistakenly interpreted as a psychiatric decompensation.

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