monitoring for symptoms of end organ damage (including gastrointestinal symptoms, chest pain and dyspnea) is recommended, and clozapine should be withdrawn if end organ damage is suspected or confirmed.

In short, we present the case of a young female who developed pleural effusion and hypereosinophilia 4 weeks after the initiation of clozapine therapy, with associated gastrointestinal symptoms. Symptoms remitted shortly upon discontinuation of clozapine. The elevated numbers of eosinophils in peripheral blood are thought to play a role in the pathogenesis of clozapine induced polyserositis.

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Psychosis as debut of sporadic Creutzfeldt–Jakob disease☆

Psicosis como inicio de enfermedad de Creutzfeldt-Jakob esporádica

Dear Editor,

Creutzfeldt–Jakob disease (CJD) is a neurodegenerative pathology belonging to the group of prion diseases or transmissible spongiform encephalopathies. It is caused by the central nervous system deposit of a pathological isoform of the normal prion protein (PrPc) present in all mammals. The mechanism by which this conformational change is produced is unknown. The accumulation of the pathological prion protein (PrPsc) gives rise to a neural degeneration that provokes a rapidly progressive fatal neurological deterioration.

There are three forms of CJD: sporadic (sCJD), familial and acquired. The sporadic form represents 85% of the cases, with greater incidence in individuals approximately 60 years old; and 90% of the patients die within a year of symptom onset, with a mean survival of 6 months.[1]

The classic clinical presentation of sCJD includes rapidly progressive dementia, myoclonus, and pyramidal, extrapyramidal and cerebellar signs. Although it is less frequent, the disease can begin with non-specific psychiatric signs and symptoms such as personality changes, behavioural changes, anxiety, depression and even as a psychotic condition, which can make initial diagnosis more difficult.[2–11]

Diagnosis is based on the clinical features and the neurological examination findings, together with the presence of alterations in the diffusion-weighted sequences (DWI) or brain magnetic resonance imaging (MRI) scan using fluid-attenuated inversion recovery (FLAIR) in the caudate nucleus and putamen or in at least two cortical regions,[12] an electroencephalogram (EEG) showing periodic sharp wave complexes superimposed on a slow background rhythm,[13] and/or cerebrospinal fluid positive for 14-3-3 protein.[1] Nevertheless, these findings are not pathognomonic and their normality does not rule out the disease. Definitive diagnosis is established using pathological studies that show spongiform degeneration, neuron loss and gliosis.[1] There is currently no cure, with treatment being merely symptomatic.

We present the case of a 53-year-old Columbian female, divorced, having a 20-year-old son. The patient had no other

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direct family members and a very limited social support network. There was no personal or familial psychiatric history. Condition began with delusions of persecution that required involuntary admission to the Acute Psychiatric Unit. Blood tests, urine analysis for drugs and cranial computed tomography (CT) scan were performed with normal results. During hospitalisation, treatment was begun with risperidone up to 6 mg/day with good response. Patient was discharged with the diagnosis of unspecified psychotic disorder and prescribed treatment with 9 mg/day paliperidone.

During outpatient mental health unit follow-up, the delusional disorder reappeared at 8 months; this was considered secondary to poor therapeutic adherence, requiring new admission to the Psychiatric Unit. This time pharmacological control of psychotic signs and symptoms was incomplete, and slowing in gait and cognitive deficits were also detected. Given the lack of familial support, she was transferred to a medium-term community health centre upon discharge and evaluation by an outpatient neurology unit was requested. The outpatient brain MRI scan showed T2 hyperintensity and FLAIR with diffusion restriction of the frontal and insular cortex, of the thalamus and of the basal ganglia, predominantly right (Fig. 1). The EEG revealed overall slowing of moderate encephalic nature.

At that time, the patient presented severe temporal and spatial disorientation, walking limitation, generalised pyramidal syndrome and myoclonus. At the functional level, there was complete loss of personal autonomy, with no language use, and the patient was confined in a wheelchair. She presented significant behavioural changes and the spinal tap could not be carried out.

Our patient was diagnosed with probable sCJD, based on internationally-agreed diagnostic criteria,[1] because she presented rapidly progressive cognitive deterioration, extrapyramidal signs, pyramidal syndrome and akinetic mutism.

It is worthwhile noting that, although psychiatric symptoms are considered rare, they are found in sCJD as prodromal symptoms; in early stages of the disease they are found in up to 40% of the cases.[14] However, presentation as a pure psychotic condition over so many months of development is not typical and the predominantly frontal alterations in our patient's diffusion sequences in the MRI scan indicate correlation with the initial signs and symptoms.

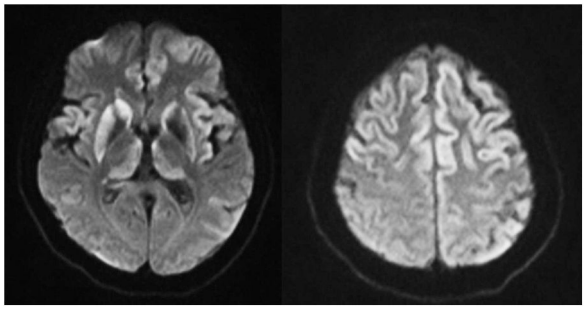

Figure 1 DWI sequences with restriction of the frontal and insular cortex, putamen and caudate (predominantly right asymmetry) and thalamus (mediodorsal nucleus).

In short, our case illustrates the need to consider CJD as a differential diagnosis in patients with psychotic symptoms or affective disorders resistant to conventional psychiatric treatment. The frequent lack of biological markers for diagnosing psychiatric illnesses can make the initial diagnosis more difficult to reach, especially in cases of atypical presentation.[15,16]

Consequently, with patients for whom there is a high level of initial CJD suspicion, but normal EEG and neuroimaging tests, repeating these analyses over the course of the disease is recommended.[14]

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Prepare the smoking cessation in severe mental illness: Early diagnosis and prevention opportunities☆

Preparar la cesación tabáquica en el trastorno mental grave: diagnóstico precoz y oportunidades de prevención

Dear Editor,

Populations that suffer severe mental disorder (SMD) face high early mortality rates. The evidence is clear: these patients have a life expectancy up to 20 years shorter and the gap between them and the general population is growing larger.[1] These figures are the result of the interaction of various factors: those characteristic of SMD itself, associated health behaviours, factors stemming from the healthcare system and related social determinants. Even so, tobacco use is the main preventable mortality factor, and 50% of the patients with SMD who cannot manage to stop smoking will die from tobacco-related causes.[2]

Together with cardiovascular diseases, respiratory processes—specifically, chronic obstructive pulmonary disease (COPD) and pneumonia—are the main causes of mortality in patients with schizophrenia or bipolar disorder (BD).[3] In spite of this, there are few studies about the onset, severity and evolution of lung damage in these patients.[4] There are currently no clinical recommendations for early diagnosis in a population that, because of a use involving higher risk (earlier start, greater dependence levels, more intense smoking) and its demonstrated impact on mortality rates, could benefit from this early diagnosis.

Developing strategies to encourage patients to enter treatment is also a high priority,[5] given that nicotine dependency can be treated safely and effectively.[6] The efficacy of multicomponent treatment programmes has been demonstrated in this population, and such programmes emphasise the importance of the preparation phase before active treatment.[7,8] To optimise motivation, it is essential to focus on certain aspects: the intensity of the intervention, the individualisation of the message and the possibility of acting on factors such as the balance between the benefits and risks of continuing to smoke. The patient with SMD usually has a poorer perception of the health risks associated with smoking and, consequently, thinks less about health benefits as a reason for making an attempt to stop.[9] Access to information on the individual tobacco-related health risks and the possibilities of preventing them can therefore represent an opportunity.

Mobile technology makes it possible to transmit health information and build motivation at any time and in any place. In treating tobacco use in the general population, even the most basic tools, such as text messages (SMS), have been extremely successful. In spite of the growing evidence as to their potential and safety in the treatment of patients with psychotic disorders, their possibilities are still unknown to a great extent in tackling tobacco use in this population.[10]

Our group is currently developing a randomised multicentre study with a year's follow-up on a sample of patients with BD and schizophrenia to establish the efficacy of a motivational intervention that offers individualised information of tobacco-related risk and the options of prevention. The level of undiagnosed lung damage—the calculation of pulmonary age and COPD presence and staging—is determined through spirometry. The intervention also explores the intensification using repeated motivational messages via mobile SMS technology.

Neurocognitive functioning will govern the reception of the information and the use that the patient might make of it. In schizophrenia and BD, deficits in several attentional functions, executive function and working memory that are essential for maintaining goal-oriented behaviour. Our design, in which these variables are controlled, should also help to ascertain their influence on motivational tools based on health information.

In short, the problem of tobacco use in this population needs new strategies that are effective in creating attempts to stop smoking and whose final goal is smoking cessation. A prevention-based intervention model is feasible and can find its ideal setting in community attention to mental health. A multidisciplinary team, one that knowledgeable about patients with SMD and their health needs, can offer a new option for approaching the tobacco-use problem by providing early diagnosis and building motivation.

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