Clinically, it is important to be aware that one patient can present different DMS and evaluate if there is any kind of hostile behavior towards the objects of the delusions.

CONFLICT OF INTERESTS

None to declare.

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Olanzapine in the management of psychosis in Huntington's disease: a case report

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Dear Editor,

Huntington's disease (HD) is a progressive autosomal dominant neurodegenerative disorder of complete penetrance, characterized by a progressive movement disorder, cognitive deterioration and various neuropsychiatric manifestations.[1] It's caused by an unstable expansion of the trinucleotide cytosine- adenine-guanine (CAG) in exon 1 of the "huntingtin" (HTT) gene located on the short arm of chromosome 4 (4p16.3).[2] The number of CAG repeats in the HD gene varies from 6 to 35 in healthy people; while those in which 27 to 35 repetitions are found could transmit the HD to their offspring.[3]

HD is a rare disease. In a recent meta-analysis, it was found that, worldwide, the prevalence of HD has been estimated at 2.71 per 100000 persons, varying in different regions: in Europe, North America and Australia a prevalence

of 5.7 per 100,000 persons has been estimated, while in Asia the estimate is 0.4 per 100,000 persons.[4]

With the aim to review the literature on neuropsychiatric manifestations in patients with HD, we present the case of a 49-year-old woman with a family history of HD, choreic movements and psychotic symptoms.

Clinical case

The patient is a 49-year-old women, divorced, with a higher technical education degree, currently unemployed. He has a family history of involuntary movements (Figure 1). In his personal history, since the 39 years, began to show irritable, at age 46 began choreic movements, being diagnosed with HD in Chile. She did not receive treatment for this disease.

Five months before admission to our service, the patient was in Chile; She lost contact with her relatives for a few weeks and later was found wandering on the public road without apparent direction. She was taken to a physician, who an aggressive, disorganized and hallucinatory behavior, in addition to the choreic movements. She is brought to Peru by her mother. In the course of the months, irritability became more noticeable. In addition, the patient refused to be taken to a health facility. The soliloquies intensified, the patient mentioned: "they wanted to kill me, they made me witchcraft".

Two months before admission, the aggressiveness of the patient was increasing, tried to smother her mother, insulted their neighbors, who also threatened to stone them because they "wanted to steal", she began undressing in public. The week prior to admission, the patient was aggressive against her mother, so they decide to take her to the "Hospital Regional de Trujillo", where she is hospitalized in the Psychiatric Service.

At the general physical examination, the neurological clinic was remarkable; there was involuntary movements of the choreic type in the extremities and neck. In the mental examination, a patient with aggressive and disorganized behavior was found; wake up, disoriented in time and space; without awareness of mental disorder; hypoprosexic; concrete and tangential thought, of self-referential and magical content, delusions of damage; dysarthric language; visual and auditory hallucinations; poor capacity for abstraction; hypoamnesia in the short and medium term; dull, irritable affect, poor impulse control; hypobulia.

In the blood analysis (blood count, liver profile, thrombotic profile, renal function, vitamin B12 and folic acid) no alterations were observed. A cerebral MRI (cMRI) was performed, which showed signs of cortico-subcortical atro-

Figure 1 | *Family pedigree of the patient. In each generation there are people affected with involuntary movements*

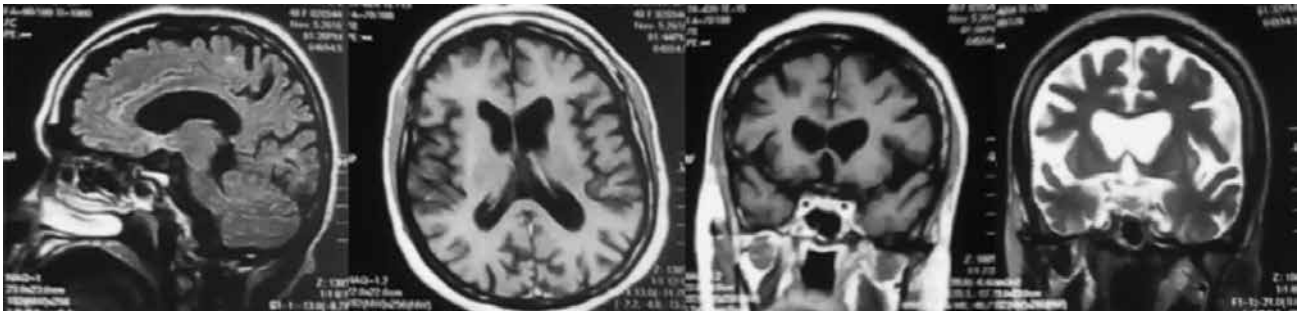

Figure 2 — *cMRI of the patient in which multiple lesions were evidenced compromising the peritrigonal white matter of both hemispheres, being hyperintense in FSE T2 and in FLAIR, signs of cortical atrophy, atrophy of caudate nucleus. The cMRI study was performed with a Toshiba Flexart 0.5T device using FSE T2 and FLAIR sequences in axial and coronal planes. Intravenous contrast material (Gadolinium) was injected*

phy with atrophy of the caudate nucleus associated with lateral ventriculomegaly (Figure 2).

The patient was diagnosed with organic psychosis (F06) secondary to Huntington's disease (G10). The treatment consisted of olanzapine 10 mg/day and sodium valproate 1,000 mg/day. The intensity of the hallucinations and delusions decreased after the fourth day of hospitalization, the aggressiveness decreased on the seventh day. After fourteen days there was no evidence of psychotic symptoms. The choreic movements began to decrease from the sixth day, becoming less evident after three weeks. At the time of hospital discharge, persistence of cognitive deterioration was evidenced.

DISCUSSION

HD belongs to the group of diseases called "rare" or "orphan". The average age of onset of symptoms is between 30 to 50 years, with a survival of 10 to 30 years, being the main causes of death in these patients, respiratory infections and suicide.[5] The clinic of HD can be divided into three major domains: 1) motor symptoms, which can be involuntary movements such as chorea, which are extrapyramidal movements, non-repetitive, arrhythmic to predominance of facial muscles, trunk and limbs, and deficiency in voluntary movements such as bradykinesia or rigidity.[5] 2) Cognitive disorders, which are inevitable, being the interindividual progression very changeable. We found deficiency in executive functions, visuospatial and perceptual deficiency, impairment in learning and problems in memory, of which many times patients are aware. As cognitive deterioration progresses, dementia could be reached.[5, 6] 3) Psychiatric disorders: depression, risk of suicide, anxiety, irritability and agitation, apathy, obsessive-compulsive behaviors, and psychosis.[5] Regarding psychiatric disorders, in a longitudinal study which investigated individuals with HD mutations in Europe between 1993 and 2011, it was found that 83% had psychiatric symptoms being apathy the main (28.1%), followed by aggressiveness-irritability (13.9%), obsessive-compulsive symptoms (13.2%), depression (12.7%) and psychosis (1.2%).[7] In Peru, a sample of 68 patients attended in the "Instituto Especializado de Ciencias Neurológicas" from 1974 to 2003, found the following motor symptoms: presence of involuntary movements 68 (100%) , problems for walking 31 (45.58%), difficulty in speaking 22 (32.35%), awkwardness in walking 7 (10.29%); whereas the psychiatric disorders were: irritability 16 (23.52%), depression 6 (8.82%), abulia 5 (7.35%), the presence of psychotic symptoms was not reported in this sample, the largest of Peruvian patients.[8] These data indicate the rarity of the presentation of psychotic symptoms in HD, which leads us to highlight the significance of the case presented. Our patient began to experience irritability at 39 years of age, a characteristic symptom of the onset of dementia associated with HD.[6, 8]

The diagnosis of HD is based on family history, clinical, genetic and neuroimaging studies. Although the definitive diagnosis of HD is genetic, the neuroimaging could provide evidence to support the clinical diagnosis, as well as for the differential diagnosis. Structural changes at different levels have been described in manifest HD: 1) Subcortical structures, in which we find atrophy of the basal ganglia, especially the striatum. Other structures as you relate are thalamus, hippocampus, pale globe, accumbens, putamen and caudate nucleus.[9] 2) Reduction of cortical thickness.[9] 3) Degeneration of the white matter.[9]

Regarding treatment, HD does not have a cure or treatment that modifies the natural course of the disease, the management is support aimed at improving the quality of life of these patients.[10] For the control of choreic symptoms, tetrabenazine, a dopamine transport blocker, is indicated.[11] In our city we do not have access to this drug, so it was decided to start treatment with olanzapine at a dose of 10 mg/day in order to attenuate choreic movements and psychosis. The scientific literature reports that olanzapine, a dopaminergic antagonist, is commonly prescribed for the treatment of motor symptoms in HD. In a study of 11 patients with HD, olanzapine was used at a dose of 5 mg, concluding that olanzapine is a potentially useful drug with significant short-term effects on the behavioral symptoms of patients with HD.[12] Another study found that high doses of olanzapine (30 mg) may be beneficial for the treatment of chorea in HD.[13] In a study of 9 patients with HD who received an average dose of 11.4 mg of olanzapine, it was concluded that this drug is an adequate alternative for patients with HD, suggesting its use in patients with severe chorea and/or presence of severe psychiatric disorders. (e.g, the psychosis described in our patient).[14]

In conclusion, as far as we know, this is the first Peruvian case of a satisfactory treatment with olanzapine in the context of a psychosis secondary to HD, being an important alternative in countries where tetrabenazine is not available.

CONFLICT OF INTERESTS

None.

FINANCING

Own.

ETHICAL ASPECTS

Both, patient and relatives gave their consents for the publication of the clinical history and images.

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