Where are the older autistic adults?: a descriptive study in adult population

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Dear editor,

In recent years, the prevalence of Autism Spectrum Disorders (ASD) have been escalating in developed countries ranging from 4-5 cases per 10,000 inhabitants to more than 1% of the general population. At present, there are data that support that the prevalence of ASD in the adult population is similar to that of infant-juvenile population[1]. The aim of this study is to describe the distribution by age and sex of adult patients diagnosed with ASD who were followed up in two specialized units.

Material and Methods

Demographic data (age and sex) were retrospectively collected for patients 18 years or over who were diagnosed with ASD and were attended during the last 12 months in the CSMA (ambulatory mental health care service) of Sant Cugat del Vallès and the Corporació Sanitària i Universitària Parc Taulí (Barcelona, Spain). The elderly population was defined as people aged 65 and over.

Results

Data for the sample stratified by age and sex is shown in Table 1. A total of 188 diagnosed cases of ASD were identified. The average age of the group is 27.4 years (SD= ±11), ages ranging from 18 to 69. At the time of data collection, the largest group of patients with ASD ranged from 18 to 25 years, which constituted 65% of the total sample. The 18 to 21 years group presented the highest number of cases. Overall, the number of people with ASD treated in our units decreased as age increased: being 69 the oldest man in the group and 53 the oldest woman. According to the data analyzed, only one case of the total population in both units would fall into the category of elderly with ASD, as the participant was older than 65. Regarding sex distribution, the number of males is higher than that of females in all age groups.

Table 1 Age and gender stratification of the adult sample with ASD

Age groups	n	%	% Men (n)
18-25	123	65.4	80% (99)
26-35	24	12.8	70.8% (17)
36-45	20	10.6	70% (14)
46-55	17	9.0	76% (13)
56-65	3	1.6	100% (3)
>65	1	0.5	100% (1)

Discussion

The data analysis reveals the lack of diagnoses of ASD in adulthood, and especially among the elderly, confirming our initial impression of the relative absence of this age group in the units that serve this population, which seems even more evident among women. There are several hypothesis that can explain this finding: 1) lack of recognition of the disorder in due time during their childhood, which in turn can be influenced by the attenuation or improvement of the symptoms and alterations of associated behavior[2]; 2) other already established diagnoses which have not been reassessed over the years[3] due to the difficulties of having retrospective diagnoses sometimes after decades; 3) lack of studies about people affected by ASD in old age leads to the persistence of ignorance about some specific or defining characteristics for the diagnosis at this stage of life, preventing its proper recognition[4]. This is especially noteworthy with regard to women, who not only are less frequently diagnosed of ASD in a 4:1 ratio, but also present greater diagnostic difficulties than men in recognizing their clinical symptoms. 4) On the other hand, the life expectancy of people affected by ASD is lower than that of the general population: it is estimated that at 65 years, hope is reduced by 3 years compared to the rest of the non- ASD population[5], a factor that may contribute to its lower presence during old age. 5) During the last stage of life, other physical and mental health problems or psychological ones, such as those related to changes in place of residence or the need for support in daily life, can easily mask the symptoms of ASD. 6) Finally, we must also consider that the higher rates of childhood detection in recent years, with the consequent establishment of specialized units can contribute notoriously to accentuate the difference in the number of patients with ASD of less and more

age within the adult stage by ensuring the link of those that are referred from infant-juvenile units to adult CSMAs.

Conclusions

It is necessary to address the identification and diagnosis of people affected by ASD in old age[6] as well as to increase the number of studies that describe the specific clinical characteristics of ASD in this age group, both with regard to inherent symptoms of the disorder as to their particular development during this stage of life. Other aspects of general health should be taken into account, as well as the assessment of the degree of adaptation/maladaptation to the particular circumstances of old age: social support and personal experience of loneliness or helplessness, among others.

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A case of comorbid Capgras and Fregoli syndromes

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Dear Editor,

Delusional misidentification syndromes (DMS) are psychopathologic phenomena in which a patient misidentifies persons, places, objects or events. The most common form of misidentification is the Capgras Syndrome (CS); other types are Fregoli, intermetamorphosis, subjective doubles, reduplicative paramnesia and autoscopic phenomena[1].

CS was first described in 1923 and includes delusions in which a familiar person or place is misperceived as being an imposter[2]. Fregoli Syndrome (FS), in which a strange person or place is misperceived as being familiar, was described in 1927[3].

DMS are more frequent than previously considered. Its prevalence in all psychiatric inpatients is 1.3-4.1%[4] and a recent study identified CS in 14,1% of first episode psychosis patients[5]. On the other hand, the co-occurrence of DMS is not common and has been a focus of attention, particularly CS and FS, respectively considered to be hipoidentification and hiperidentification phenomena[6] (also known as delusions of hypo- and hyperfamiliarity[7]).

We aim to present one case of co-occurrence of Capgras and Fregoli syndromes and review the previously reported cases.

Case Report

This is the case of a 52-year-old unemployed black male, born at Guinea-Bissau but residing in Portugal since 1986, with his spouse and two children.

The patient was taken to the emergency service by the police, due to behavior disorder, abnormal speech and verbal aggressiveness towards family members.

His spouse described strange behaviors occurring for eleven months, watching only Nazi themed films and locking all the windows and doors. In the past two months this

behavior aggravated, with distrust towards the spouse once he believed, she had been substituted for a Nazi general. He started locking his daughters' room at night and being very protective of them, fearing the general who has substituted his wife. Furthermore, the patient started to believe that an anonymous man he saw on television was chasing him, pointing to different people believing they were him.

At the psychiatric emergency evaluation, he presented high levels of anguish and the persecutory, reference, Capgras and Fregoli delusions described above were confirmed. Hallucinatory activity, first-rank symptoms of schizophrenia and blunted affects were excluded.

He was admitted to the inpatient ward. Medical history had no relevant data and toxic substances use was excluded. Analytics, cerebral CAT scan, electroencephalogram and psychological evaluation (IQ included) showed no abnormalities.

The patient's personal history had several potential traumatic events: he was abandoned by his mother at age 6; lived in a region with frequent warlike conflicts for years; in Portugal, he was victim of racial discrimination, even by his wife's family; he lost his job, resulting in financial difficulties. His wife described him, previously to the symptoms, as a calm and very social person, who helped a lot of people, although he was always apprehensive and frequently insurgent respecting discrimination and political issues.

He was initially medicated with risperidone (up to 6 mg/day) with no response and, subsequently, medicated with haloperidol (up to 10 mg/day). A significant improvement was observed with resolution of the misidentification delusions, although persecutory ideas were not totally extinct. His behavior and interaction with his wife improved substantially. He was diagnosed with Persistent Delusion Disorder. One year after discharge, the patient is followed at the outpatient clinic, medicated with haloperidol decanoate (50 mg/month), and is psychopathologically stabilized.

Discussion

The co-occurrence of Capgras and Fregoli delusions is not common. Few cases have been published in literature: a 34-year-old male with paranoid disorder and borderline intellectual functioning, who presented with Capgras, Fregoli, intermetamorphosis and subjective doubles syndromes[8]; a 37-year-old male with schizophrenia, Capgras, Fregoli and intermetamorphosis syndromes[9]; a 41-year-old male with CS and FS[10]; a 58-year-old male with paranoid psychosis and cognitive impairment who presented with CS, FS, Cotard and Koro delusions and a *folie a deux*[11]; three females of 36, 19 and 62 years old with schizophrenia and CS and FS[12-14]; a case of coexistence of Capgras, Fregoli, and Cotard's syndromes in a 14-year-old girl[15]; a case of combined erotomania, *folie a deux*, nihilistic delusion, and Capgras and Fregoli delusions in a 22-year-old male with schizophrenia[16]; and a 43-year-old male with schizophrenia, Fregoli, Capgras and subjective doubles syndromes[17].

None of the cases described a Persistent Delusion Disorder, as the herein reported case. DMS can occur isolated, but are most commonly associated with psychiatric disorders (mainly, schizophrenia and mood disorders)[6] and with focal and diffuse neurologic conditions usually involving right hemisphere and frontal lobes lesions[7].

DMS have been associated with violent behavior[18]. In this case, the patient presented hostility towards the double (his wife) but didn't perpetrated any physical aggression. A recent study associated violent acts in DMS with specific delusions, including megalomania, persecution, negative affect and identified targets[18], almost all present in the case reported.

Initially, DMS were explained by psychodynamic theories, that considered the syndrome as a way of dealing with ambivalent feelings towards a subject, usually transferring the feelings of anger to the "impostor", in order to maintain only positive feelings for the beloved subject[8].

In subsequent studies, neuropsychological deficits were reported involving different cognitive domains (memory, executive functioning, and visuospatial processing)[7].

Different theories have been proposed, involving abnormalities in facial recognition systems. One model postulates that, in CS, patients present with a dissociation between the "face recognition units" (representations of familiar people's faces) and the normal autonomic reaction to a familiar face[19]. Therefore, CS would result from the experience of lacking emotional response to a familiar person, creating a new meaning for this situation. Another factor was proposed to justify the delusion itself: a failure of the "system of evaluation of convictions" or in abductive inference processes[6]. Concerning FS, a dissociation between the "person identity nodes" (familiar people's personal information) and the "cognitive system" was proposed. Thereby, the "cognitive system" overexcites certain "person identity nodes", in a way that one specific node would be sufficient to identify a familiar person, even if it's someone else[19]. Currently, there is no consensus about the mechanisms underlying these phenomena.

Conclusions

With the increasingly reported incidence of DMS and co-occurrence of hypo- and hyperfamiliarity syndromes, it is imperative to clarify the psychopathologic mechanisms and etiologic factors of these syndromes.

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*

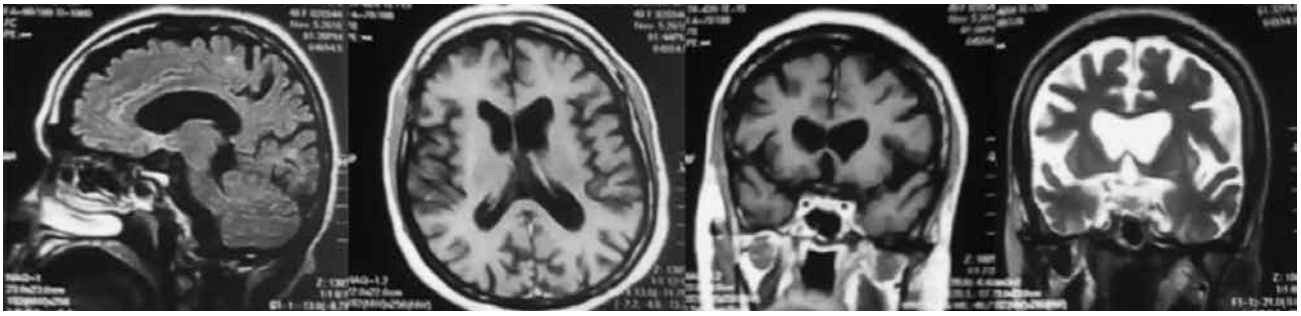

Figure 2 — *cMRI of the patient in which multiple lesions were evidenced compromising the peritrigonal white matter of both hemispheres, being hyperintense in FSE T2 and in FLAIR, signs of cortical atrophy, atrophy of caudate nucleus. The cMRI study was performed with a Toshiba Flexart 0.5T device using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