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Asperger's disorder in adulthood: a case report

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Asperger's disorder is a pervasive development disorder. It involves qualitative disorders in social relationship and communication as well as restricted and repetitive interests and activities, with no delay in language acquisition. Although Asperger's disorder is an illness that begins in childhood, its diagnosis may frequently not be done until later stages.

The case presented is about a 21 year old man with a diagnosis of schizoaffective disorder who, after several admissions, was sent to the Rehabilitation Hospital Unit for stabilization and diagnostic study given the atypical features of his case. The psychopathological examination showed disorders in social relationships, psychomotricity and communications that had begun in his childhood. All these data, and the results of the biomedical and psychological diagnostic tests oriented us towards the presence of a dual diagnosis of Asperger's disorder and schizoaffective disorder.

The presence of common symptoms between the AD and other psychiatric diseases as well as the possible existence of comorbidity may lead to an incorrect or late diagnosis.

Key words:
Comorbility. Differential diagnosis. Asperger's disorder.

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Síndrome de Asperger en la edad adulta: a propósito de un caso

El síndrome de Asperger (SA) es un trastorno generalizado del desarrollo que implica alteraciones cualitativas de la comunicación, de la interacción social e intereses y actividades restringidos y repetitivos, sin retraso en la adquisición del lenguaje. A pesar de que es un trastorno que se inicia en la infancia, el diagnóstico puede no realizarse hasta etapas posteriores.

El caso corresponde a un varón de 21 años, diagnosticado de trastorno esquizoafectivo (TE), que es derivado a la Unidad Hospitalaria de Rehabilitación (UHR) para estudio diagnóstico dadas las características atípicas del cuadro. La exploración psicopatológica evidencia alteraciones en el área de las relaciones sociales, psicomotricidad y comunicación iniciadas en la infancia. Estos datos, junto con los resultados de las pruebas biomédicas y psicodiagnósticas realizadas, orientan hacia la existencia de un diagnóstico dual de SA y TE.

La presencia de síntomas comunes entre SA y otras enfermedades psiquiátricas, así como la posible existencia de comorbilidad, puede llevar a un diagnóstico tardío o erróneo.

Palabras clave:
Comorbilidad. Diagnóstico diferencial. Síndrome de Asperger.

INTRODUCTION

The term «autistic» was first used by E. Bleuler (1911) to describe a symptom of schizophrenia: social isolation.

Leo Kanner (1943) described a common behavior pattern in children, that he called «early infantile autism». H. Asperger (1944) describes the behavior of a group of children characterized by being socially rare, having an adequate but pedantic speech, maintaining interests circumscript to specific subjects, poor non-verbal communication, alterations in psychometricity and normal intelligence but with learning difficulties[1].

Between the years 1930 and 1970 there was a «unitary» view of infantile psychoses that included the concepts of autism and infantile schizophrenia. The need to differentiate these disorders was pointed out after the 1970's. Autism was included for the first time in American nosology in the third edition of the DSM (1980), within deep development disorders, but it was not until the DSM-IV (1995) when generalized development disorders (GDD) and the asperger syndrome (AS) would occupy their own diagnostic and differentiated category of autism.

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Information on the prevalence of AS is limited, proportions of 3.7 to 7.1 per 1,000 children and 7 to 16 per 10,000 in autism being mentioned[2].

Even though this disorder begins in childhood, it may not be diagnosed until its later stages, after years of trekking through different resources and professionals. Different factors may contribute to late and/or incorrect diagnoses: lack of quantitative speech disorders, comorbidity with other psychiatric diseases (affective D., OBD, psychotic D., sleep D., etc.); the need to observe diagnostic criteria from the third year of life may entail reliability problems; sharing a high number of symptoms (preservations, psychomotor restlessness, irritability, etc.) with other psychiatric entities, existence of somatic diseases (tuberous sclerosis, fragile X syndrome, moderate-severe sensorial alterations, etc.) where similar symptoms may appear.

The principal confounding areas when making the diagnosis are established with high functioning autism (HFA), infantile schizophrenia and schizoid personality disorder, that would be essential to establish a differential diagnosis.

In the bibliography, there is a continuous debate about HFA and AS, focused on the fact that AS may be a different disorder than the HFA or a continuum within the autistic spectrum.

Asperger (1944) studied its differentiation with infantile schizophrenia. He indicated that the personality patterns of children who developed schizophrenia were different from children with «autistic psychopathy». There is no unanimity on its differentiation with schizoid disorders. While some authors find clear differences between both entities, others[5] speak of unifying them under the same category.

CLINICAL CASE

A 21 year old male who had been adopted was referred for restrained admission to the RHU in the acute unit (AU) due to behavioral and heteroaggressive disorders with the family.

The psychopathic examination revealed that he was self- and allopsychically oriented as well as a conscious and collaborating patient. Low, fluctuating mood, feelings of incapacity and effective ambivalence towards the family. Intermittent eye contact, non-expressive facial expressions. He showed no awareness of the difficulties and minimized attitudes. His speech was hypoprosodic, fluent but repetitive and sometimes had empty content. He was easily distracted, with altered abstract thinking, preservation and motor stereotypes. Distrustful, without showing delusional ideation. He denied sensorial-perceptive alterations, although he admitted having had them in the past. Sleep and appetite were conserved. There were special physical characteristics, with rough and prominent traits as well as motor clumsiness.

No drug allergies or toxic habits. Medical background: phimosis operation and appendectomy. The physical examination was normal. Neurological examination: conserved upper functions, no meningeal signs, cranial pairs present and symmetric, conserved mobility and sensitivity. Poor motor coordination.

Pathobiography

His parents reported he had learning and interaction difficulties since his early childhood. He had no psychomotor developmental delay although he was clumsy. He began to walk at one year of age, had a strange way of running, difficulty dressing, and tying his shoes. Speech development was normal. At six years, relationship difficulties increased. He was shy and had tics in his hands. He began to read early and his capacity to retain data stood out. His first visit to the child-adolescent mental health site (CA-MHS) was at 11 years old due to shyness, behavioral disorganization, relaxation problems and low self-esteem. Up to then he had had good scholastic performance and no conflicts at home. His second visit to the CA-MHS at 14 years of age was due to dysphoria, aggressiveness, increased impulsiveness, multiple motor tics, behavioral problems at home and at school and regressive behaviors. The Wechsler Intelligence Scale for children was administered, showing a total IQ of 98, and the chromosomic study was performed to rule out fragile X syndrome. During his adolescence, he was socially isolated and interested in mystic and paranormal subjects, auditory hallucinations appearing for the first time.

At 18 years of age, follow-up was begun in the mental health site (MHS), where he collaborated little with no awareness of his limitations. Affective instability, above all in spring and fall, and mystic and paranoid psychiatric symptoms against the family were seen, and he was diagnosed of schizophrenic disorder. No continuous follow-up was done. He rejected treatment, and the behavior disorders intensified. At 21 years of age, he was admitted in the AU due to suicidal ideation, escaping from home, decreased intake, physical and verbal heteroaggressiveness and soliloquies. When admitted, he was conscious and oriented, with speaking pressure and stuttering; tangential speech, self-referentiality with the family, feelings of emptiness, hypothymia, apathy and insomnia. No sensorial-perceptive disorders were observed. He began neuroleptic and anxiolytic treatment with improvement of the mood status although the self-referentiality persisted. After stopping treatment, he was admitted again with similar symptoms, the diagnosis of SD being maintained (DSM-IV).

Giving his torpid course and the atypical characteristics of the picture, he was referred to the RHU for differential diagnosis and clinical stabilization. Biomedical and psychodiagnostic examinations were conducted that contributed to establish a differential diagnosis.

Complementary examinations

Psychodiagnostic tests

Autism diagnostic interview-revised (ADI-R)[6] (table 1)

Difficulties were observed in the four areas included in the ADI-R:

- *Qualitative incapacity in reciprocal social interaction.* Incapacity to use non-verbal behaviors in the regulation of social interaction (the patient does not maintain visual contact and has no social smile). Imaginative playing does not appear with peers or interest for other children: lack of shared interests and social-emotional reciprocity.
- *Qualitative incapacity in communication.* Difficulties in social conversation (limited to exchange of information); pronominal inversion and lack of social imitative play (does not imitate the actions of others).
- *Restricted, repetitive and stereotyped behavior patterns.* The presence of circumscribed interests (data accumulation), rituals, inflexible customs and repeated use of objects.
- Abnormality or deviation in evident development before 36 months of age.

Table 1 Results on the Wechsler adult intelligence scale (WAIS-III) and on the autism diagnostic interview (ADI-R) dimensions

WAIS-III	Score
Intelligence quotient	
Verbal	105
Manipulative	80
Total	93*
Indexes	
Verbal comprehension	109
Perceptional organization	83
Working memory	106
Processing speed	86
ADI-R	**Score****
Qualitative incapacity in reciprocal social interaction	27/30
Qualitative incapacity in communications	16/26
Restricted, repetitive and stereotyped behaviors	5/16
Abnormality or deviation in the evident development before 36 m of age	4/5

* This serves as an approximate value, since it should not be calculated due to the important discrepancy between verbal and manipulative.
** Direct score/maximum score.

Neuropsychological examination

A battery of cognitive tests was done. These included: Wechsler adult intelligence scale (WAIS-III) (table 1), Wisconsin card sorting test (WCST) and graphic and verbal tests for the evaluation of the Theory of Mind.

Verbal IQ was located in normal-high functioning level while manipulative IQ was in a normal-low one, there being a significant and unusual difference between them in regards to their standard type group. Significant differences were observed between verbal comprehension-perceptional organization, perceptional organization-working memory and working memory, processing speed, indicators of the presence of difficulties in visual-spatial and visual-motor reasoning. Among the verbal subtests, a lower score was obtained in understanding, indicating difficulties in the understanding of relationships and social judgments.

His deterioration level was greater than the mean on the WCST due to the low percentage of responses on the conceptual level. This indicated comprehension difficulties of the strategy necessary to establish categories.

Difficulties were also observed in tests that evaluated the theory of mind, where he responded from his point of view, being incapable of putting himself in someone else's shoes.

Biomedical examinations

Tests were conducted in order to rule out other possible diagnoses: phenylcetonuria, measles embryopathy, tuberous sclerosis, herpes encephalitis, hydrocephaly, fragile X syndrome, lactic acidosis, purine disorder, sensorial, visual and auditory disorders and moebius syndrome.

Complete blood count and biochemistry normal, serologies negative for: HIV, HCV, HBV and LUES. Chest X-ray and ECG without outstanding findings. Normal Brain CT scan. EEG with mildly irregular activity, without focalities or irritative paroxysms. Otorhinolaryngological (audiometry) and ophthalmologic (eye fundus) examinations normal.

The information obtained in the clinical interviews with the patient and his parents, data supplied by the CA-MHS (alterations initiated in early childhood and manipulative difficulties in spite of their being an IQ within normality), existence of previous normal cariotype, direct and continued observation in our unit (difficulties in social interaction and psychomotricity) and results of the complementary examination conducted, have led us to suggest the presence of GDD/AS[7], comorbid with ST. During his admission, drug treatment was adjusted and the areas of difficulty were ap-

proached through group activities and individual therapy. On discharge, orientation was directed towards the search for specific resources and to continue check-ups in the reference MH site.

CONCLUSIONS

We present the case of a male diagnosed of GDD/AS and comorbid schizoid disorder, the latter appearing at the beginning of the adults age which was added to the difficulties already initiated in childhood by AS. Asperger commented that only one out of every 200 cases with AS finally developed symptoms of the psychotic spectrum. More recent studies indicate that comorbidity between AS and psychosis is low, the risk of having psychotic symptoms being low among persons with AS[8]. In our case, the fact that the patient had periods of affective instability and psychotic type symptoms justifies the maintenance of this diagnosis. Since AS shares a high number of symptoms with other more prevalent psychiatric pictures, it may be diagnosed erroneously[8]. This fact, together with the possible comorbid existence of another psychiatric disease, become factors that may contribute to a late diagnosis of AS, as in the case which we present. Recent research indicates that AS has a greater prevalence than thought[9]. Evidence has been found that suggests that there may be an early history orienting towards the diagnosis of GDD among children diagnosed of childhood psychosis[10].

Diagnosis of GDD is complex and requires the participation of different clinical professionals to propose the required examinations and complementary tests in a coordinated way.

It is recommended to include the following in the biomedical examination: physical and neurological examination, EEG, ophtalmological examination (eye fundus and/or visual evoked potentials) and otorhinolaryngological (audiometry and/or evoked potentials) as well as complementary tests, such as blood analyses, genetic studies and metabolic studies. There are no genetic, biochemical markers or imaging techniques that provide data leading to the clinical diagnosis.

Knowledge on AS has increased in the last decades, leading to an increase in demand for care by these subjects in the healthcare services. Specific analysis techniques and evaluation that make it possible to assess the personal background, current symptoms, areas of difficulty and adaptive behavior need to be developed to contribute to an early diagnosis. In the same way, creation of specific resources to treat this disorder will be essential for the patient's evolution and integration within the community.

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