hipótese foi de intoxicação por drogas, uma vez que é comum em nosso país o tráfico feito por estrangeiros procedentes da África. A possível jargonofasia, que poderia até ser confundida com alteração da linguagem secundária a acidente vascular cerebral, logo foi descartada com a história fornecida pelos acompanhantes. Essa história evidenciou um fator estressor importante (distância e medo de ser traída) associado à dificuldade de comunicação (apenas a paciente, entre as pessoas que a acompanhavam, não falava o português).

Aspectos relevantes para o diagnóstico de um transtorno dissociativo teriam sido facilmente identificados se as particularidades socioculturais tivessem sido observadas desde o início da abordagem. Jureidini conceituou a dissociação como um estado de alteração da consciência, no qual as barreiras normais entre memórias conscientes e inconscientes, desejos e crenças, são quebradas, enquanto barreiras amnésticas vêm à tona.[2] Temos aqui uma alteração funcional de uma paciente com fator estressor identificável e sem comprometimento anatômico que a justificasse, lembrando a importância da evolução histórica do diagnóstico dos transtornos dissociativos.[3] É importante evidenciar que muitos estudos reforçam a idéia de que a cultura exerce uma grande influência na apresentação e determinação dos sintomas, principalmente psiquiátricos.[4] Todos os médicos devem estar atentos e respeitar as diferentes formas de seus pacientes demonstrarem seus sintomas.

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Tardive dystonia, a case report

Distonia tardia, um relato de caso

Dear Editor,

Meige's syndrome II/Brueghel's syndrome is a disabling spasm of the facial musculature consisting of primary blepharospasm followed by abnormal facial movement as yawning, jaw opening, and abnormal tongue movements.[1] We describe a 54-year-old man, whose delivery had been assisted by forceps. He was diagnosed with persistent delusion disorder in 1991 and began treatment with a combination of periciazine (up to 25 mg/day) and biperiden (2 mg/day). This treatment continued until 1995, when the patient began to complain of diurnal bruxism. This condition gradually worsened and, as a consequence, he cracked some teeth. A few months later blepharospasm began, followed by anterior neck spasm. In 1996, the patient began using risperidone (2 mg/day) and reported improvement of motor symptoms. The blepharo and neck spasms returned in 1998, and clozapine was prescribed. The patient reported improvement in doses of up to 300 mg/day. A year later, due to financial difficulties, this drug was suspended and he continued treatment with sulpiride (400 mg/day). In the next two years the dystonic movements worsened progressively due to the use of this medication, and involuntary tongue protrusion started. Severe speech impairment lead this patient to social reclusion and retirement, which was aggravated by the incapability to drive and frequent falls wile walking due to the visual impairment of the blepharospasm. In 2004, clozapine was restarted (100 mg/day) and combined with clonazepam (4 mg/day), resulting in an important improvement of the blepharo and neck spasm, but tongue protrusion persisted. Botulinum toxin was applied around the eyes and in the tongue. After the first application there was complete blepharospasm remission, although there was still a little unilateral ptosis and only a mild reduction of tongue protrusion. Four months later, after the second application, the result was a total remission of the blepharospasm with no ptosis, and an important partial remission of tongue protrusion. During the one-year follow-up the patient continued with the same difficulty in spoken articulation, but reported a gradual decrease in social limitations.

In our case, tardive dystonia (TD) began insidiously and progressed over years until it became static. TD runs a chronic course and spontaneous remission is uncommon even if the antipsychotics are discontinued.[2] TD also causes pain, physical and emotional disability as seen in this case.

Besides exposure to antipsychotics, other important risk factors for tardive dystonia in this case were a possible history of head injury at birth and male gender.[3] Some cases of TD may represent late-onset congenital torsion dystonias or delayed-onset dystonia secondary to prenatal injury provoked or unmasked by antipsychotics.[4]

Clozapine has been found useful in TD, especially because of its anti-D1 action [2]. Lieberman et al. reported 43% improvement in 30 patients treated with clozapine.[5] Treatment with botulinum toxin is justifiable in refractory patients. Tarsy et al. reported, in a series of 38 affected body regions among 34 patients, that 29 were moderately to markedly improved by botulinum toxin type A injections.[6]

In this case, social limitations of daily living and interaction caused by dystonic movements were a more severe impediment than the primary disease. This movement disorder seems to draw a progressive and independent course, in spite of the interruption of typical neuroleptics or the use of atypicals. Best results were obtained with the continued use of botulinum toxin.

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Obsessive-compulsive symptoms in non-active rheumatic fever

Sintomas obsessivo-compulsivos em febre reumática inativa

Dear Editor,

Rheumatic fever (RF) is an autoimmune disorder that follows infection by specific strains of β-hemolytic streptococci.[1] Obsessive-compulsive symptoms (OCS) were first described in Sydenham's chorea (SC), the late central nervous system (CNS) expression of RF. In the last 10 years, consistent reports have found higher frequencies of obsessive-compulsive disorder (OCD), OCS and tic disorders (TD) in prepubertal RF children with[1] and without[2-3] SC. Swedo and colleagues called these neuropsychiatric disorders subgroup by the acronym PANDAS,[3] whose validity as an independent entity has been largely discussed. The association between RF and OCD spectrum symptoms has been well documented in the active phase of RF[2-3] and has been hypothetically attributed to antibodies directed against invading bacteria that cross-react with basal ganglia structures.[3] Therefore, we decided to further explore the relationship of RF and OCD spectrum symptoms in patients in the non-active phase of RF.

A descriptive study performed in an academic Brazilian hospital - *Oswaldo Cruz Hospital* (Recife-PE) - included 50 consecutive adult patients from the outpatient clinic for valve heart disease, who have had at least one acute RF episode in the past. Exclusion criteria were, age under 18 and over 45 years and/or the presence of neurological disorders or other heart diseases of non-rheumatic etiology. Written informed consent was obtained. The RF diagnosis was established according to modified Jones criteria.[1] Psychiatric symptoms were assessed by SCID-IV and diagnosis of OCS was made when patients met criteria only for obsessions or compulsions.[4] *Best Estimate Diagnosis*[4] was made with two senior psychiatrists (ECM; KP). Our sample comprised 14 men and 36 women with a mean age of 34.5 years (SD = 9.8). We found OCS as the most frequently reported psychiatric symptom occurring in 16 patients (32%). Agoraphobia was reported in 1 individual (2%), generalized anxiety disorder in 5 (10%) and major depressive disorder in 14 (28%). Only 1 individual (2%) presented OCD in its complete form. The OCS was not related to the number of lifetime RF exacerbation episodes (p = 0.16) or previous cardiac surgery (p = 0.37).

We recently reported the presence of OCD, TD and body dysmorphic disorder in another sample of non-active RF.[2] Furthermore, in a recent controlled study performed in another academic hospital - *InCor HCFMUSP* (Sao Paulo-SP) - assessing 97 heart disease outpatient subjects we found increased frequencies of OCS in non-active RF compared to controls (p = .02).[5]

Thus, we have systematically found OCD spectrum symptoms in different samples of RF patients in the non-active phase, which is theoretically interesting and intriguing, as chronic sequels that continue to develop long after the initial RF episode have been described concerning joints, cardiac tissue, and the CNS[1-2,5] in the form of chorea. However, it is possible that RF acute changes could have persisted or triggered other immunologic responses. The low prevalence of full OCD and the absence of a control group are important limitations of these studies. Further neuroimmunological and genetic studies are needed to elucidate the mechanisms through which active and non-active RF confers a high risk for these neuropsychiatric symptoms.

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