(protein coding regions).[4] The silent polymorphism 1287 A/G (rs5569), located in the exon 9, has been previously studied in other psychiatric disorders, such as schizophrenia, bipolar disorder, major depression, Tourette syndrome and panic disorder. These investigations produced inconclusive results so far.

The present study investigated the 1287 A/G variant (rs5569) in 75 DSM-IV OCD patients and 317 matched healthy controls. This study was previously approved by the Clinical Hospital Ethics Committee and all participants gave their written informed consent. Genomic DNA was extracted from venous blood samples and the exonic silent polymorphism (1287 A/G – rs5569) was analyzed as described by Jönsson et al.[5] PCR products were resolved on 2% agarose gels and visualized by ultra-violate light after ethidium bromide staining. We used a chi-square test for the analysis of the differences of allele and genotype frequencies between OCD patients and controls.

The genotypic distributions were in Hardy-Weinberg equilibrium. We did not find differences in the allelic ($OR = 1.14$ $0.74 < OR < 1.75$; $X^2 = 0.38$; 1 d.f.; $p = 0.53$) and genotypic ($X^2 = 0.51$; 2 d.f.; $p = 0.77$) distributions (Table 1). Our results do not support the association between the 1287 A/G polymorphism in the NET gene with OCD in our Brazilian sample.

Table 1 – Allelic and genotyoic distribution of the norepinephrine transporter gene

	OCD	Controls	X^2	p value
ALLELES				
A	113 (75.33)	462 (72.87)	0.38	0.53
G	37 (24.66)	172 (27.12)		
Total	150 (100)	634 (100)		
GENOTYPES				
AA	42 (56.00)	169 (53.31)	0.51	0.77
AG	29 (38.66)	124 (39.11)		
GG	4 (5.33)	24 (7.57)		
Total	75 (100)	317 (100)		

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Importância dos aspectos culturais em um caso com agitação psicomotora

Cultural aspects importance in a case with psychomotor agitation

Sr. Editor,

A globalização tem derrubado as fronteiras mundiais. Tem colocado os médicos da atualidade em contato mais freqüente com pacientes de diferentes etnias e culturas. Segundo Konner, diferentes atitudes, conhecimentos e condutas são necessários para a prática clínica neste novo século.[1] Em virtude desse fato passou-se a dar importância a psiquiatria transcultural, que pode fornecer as ferramentas para diagnóstico e tratamento efetivo através da remoção das barreiras culturais e lingüísticas.[1] Relatamos o caso de uma paciente de 32 anos, casada, com prendas domésticas, natural e procedente de Menongue, Angola. A paciente foi levada por amigos ao Pronto Socorro da Santa Casa de Misericórdia de São Paulo após um período de 6 horas de alteração de comportamento. Segundo os acompanhantes, ela teria sido possuída pelo demônio, tornando-se muda e isolada, evoluindo posteriormente com agitação e estado de transe. Durante avaliação inicial, a paciente foi colocada no leito, adotando posturas de contorção corporal e revirando os olhos para cima. Passou a falar de modo rápido e incompreensível, o que foi inicialmente interpretado como jargonofasia. No entanto, logo foi constatado que se tratava do dialeto de uma região do país de origem. O exame físico não apresentava alterações. Os exames laboratoriais eram normais. Foi realizada radiografia de abdômen no intuito de encontrar algum corpo estranho (como porte de drogas), porém sem achados. Os acompanhantes informaram que a paciente, apesar de morar em Angola, não falava português, pois esta língua é um diferencial socioeconômico naquele país e a ela provinha de uma classe social desfavorecida. Estava no Brasil há dois dias, nunca havia ficado longe do marido e demonstrava grande preocupação pelo fato dele ter outras mulheres. Foi medicada com 2,5 mg de haloperidol, com melhora significativa dos sintomas após 1 hora, quando referiu não saber o que havia acontecido. Os acompanhantes descreveram outros episódios semelhantes no passado, todos em situações de estresse.

O presente caso evidencia a necessidade da equipe médica observar aspectos transculturais na doença mental. Foi possível observar uma paciente, recém chegada ao Brasil, admitida no Pronto Socorro com quadro de agitação. A primeira

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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