Cartas aos editores

Organic vs. psychogenic tics

Dear Editor,

We would like to comment on Mejia and Jankovic's paper: "*Secondary Tics and Tourettism*". First, it is remarkable that secondary tics were twice as prevalent in males (65.1%) as in females (34.8%). The higher prevalence of Tourette's syndrome (TS) in males is well-known, and this study could point to a greater susceptibility of males to develop tics be the cause TS or not.

Second, some points remained unclear, such as the criteria for diagnosing psychogenic tics. As the rate of 10.3% is quite high, the criteria used should have been specified. Others have documented psychogenic movement disorders (PMD) prevalence rates in about 3.3% of the cases, but tics were rarely found in their samples.[1]

The cited reference[2] provides a diagnostic guideline for PMD such as dystonia, tremor, paroxistic dyskinesia, parkinsonism, gait disorders, hemifacial spasm, spasmodic dysphonia, and myoclonia, but has not included psychogenic tics.

Fahn and Williams classified patients in four categories (documented, clinically established, probable and possible) based on the level of certainty of having psychogenic dystonia. This classification is often used for other movement disorders. They define *documented PMD* as involuntary movements which are persistently relieved by psychotherapy, suggestion, placebo, or the patient is witnessed as being free from the involuntary movements when supposedly unobserved; and *clinically established PMD* are movements which are inconsistent or incongruent with classic descriptions of the acknowledged movement disorder, associated with other psychogenic signs on neurological examination, multiple somatizations, or obvious psychiatric disturbance. The *probable* and *possible* categories have less clinical evidence being often excluded from most studies.[3]

Thomas and Jankovic[2] describe indicators of presence of PMD including abrupt onset with disability soon or immediately after onset; response to placebo or suggestion, selective disability, dramatic resolution, increase with attention and cessation with distraction. Those criteria may not differentiate organic from psychogenic tics. Tic disorders usually have a waxing and waning course, may begin abruptly and with maximal severity. Tics may also be under some voluntary control, thus, temporary suppression of symptoms may often occur. Their intensity may also vary with attention (usually decreasing with distraction), a differential feature of psychogenic tremor that may not be valid for tics.[2]

Organic movement disorders are more frequently misdiagnosed as psychogenic than the opposite,[4] and their coexistence is very common.[1] The current diagnostic criteria used for PMD are not definitive and should be considered rather as a useful guide for clinical investigation. Conversive disorder should be considered after the exclusion of neurological diagnoses.

The high frequency of psychogenic tics in this sample indicates the need of well-defined criteria to characterize psychogenic tics. This dichotomy of organic versus psychogenic tics remounts to Charcot's era, when Gilles de La Tourette's syndrome or *maladie* des tics at that time, was considered a heredo-degenerative disease, contrarily to hysteria and chorea (the supposed psychogenic tics). However, as "organic" tics may begin or aggravate due to stressor events, we emphasize the need of careful assessment and development of rigid criteria for psychogenic tic disorder diagnosis.

Aline Sampaio, Ana G Hounie
Projeto Transtorno do Espectro Obssessivo-Compulsivo (PROTOC) do Instituto de Psiquiatria do Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo (FMUSP)

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On Secondary Tics and Tourettism

Dear Editor,

We kindly thank Drs. Hounie and Sampaio for their interest in our recent paper on "Secondary tics and tourettism".[1] As they mentioned, our sample of 155 patients with tics and co-existent disorders included 101 (65.1%) male and 54 female (34.8%) patients. Although a higher prevalence of Tourette syndrome (TS) in males has been well recognized,[2] we consider the higher proportion of male patients in our population of patients with secondary tics coincidental and do not believe that our study provides sufficient data to point to a greater susceptibility of males to develop secondary tics. Nonetheless, we believe that the study of risk factors associated with the development of secondary tics, including gender, may play an important role in understanding the physiopathology of tics and TS.

We would also like to clarify that the 16 (10.3%) patients found to develop psychogenic tics in our series of 155 patients were diagnosed using previously published criteria.[3] In all our

patients, the existence of tics could not be directly attributed to a lesion or dysfunction of the nervous system, and was derived in all cases from psychological or psychiatric causes. We understand Drs. Hounie and Sampaio's concern of the current use of diagnostic criteria for all psychogenic movement disorders, and encourage the scientific community to use the available criteria[3-4] as a guide to the clinical assessment of their patients.

Nicte I Mejia, Joseph Jankovic
Parkinson's Disease Center and Movement Disorders Clinic, Department of Neurology, Baylor College of Medicine, Houston, Texas, USA

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Cardiomiopatia em paciente tratada com clozapina

Sr. Editor,

A prevalência de esquizofrenia é de aproximadamente 1% na população em geral e de 30 a 60% destes pacientes não respondem ao tratamento com antipsicóticos típicos. A clozapina – antipsicótico atípico destacado no tratamento de pacientes refratários[1-3] – tem sido associada com baixa ocorrência de sintomas extrapiramidais, alteração de condução cardíaca, aumento de prolactina e síndrome neuroléptica maligna. Entretanto, podem ocorrer efeitos colaterais potencialmente fatais como agranulocitose.[1] Relatos de miocardite com cardiomiopatia e insuficiência cardíaca fatal sugerem um aumento do risco de 17 a 322 vezes em pacientes tratados com clozapina. O risco absoluto é estimado entre 1:500 pacientes tratados a 1:10.000 pacientes tratados.[4] Este efeito parece estar associado à resposta auto-imune mediada pela Ig-E, devido aos relatos de eosinofilia nestes quadros clínicos.[4-5]

A seguir, descrevemos um caso ilustrativo desta possível complicação no tratamento com clozapina. Paciente do sexo feminino, 20 anos, com diagnóstico de Esquizofrenia Paranóide desde os 16 anos, internada em unidade psiquiátrica, quatro internações psiquiátricas prévias, em uso de risperidona, ácido valpróico, carbonato de lítio e clonazepam. Já havia recebido haloperidol e clorpromazina em internações anteriores. Persistia com alucinações auditivas, delírios místicos e paranóides, além de hetero-agressividade. Foi considerado o diagnóstico de esquizofrenia refratária e indicado o uso de clozapina. Apresentava enzimas hepáticas, hemograma e eletrocardiograma (ECG) normais. Foi iniciada clozapina gradativamente associada à diminuição progressiva das outras medicações. No 13º dia de uso da medicação, a paciente apresentou hipotensão postural, dispnéia, hipoxemia e hipertermia (temperatura axilar de 39°C). Evoluiu com freqüência respiratória de 38 movimentos respiratórios por minuto, taquiarritmia ventricular (freqüência cardíaca de 260 batimentos por minuto), sendo transferida ao Centro de Tratamento Intensivo (CTI). Foi realizado ecocardiograma que demonstrou paredes cardíacas com espessura normal e hipocinesia difusa, acarretando comprometimento da função sistólica global, sendo a fração de ejeção do ventrículo esquerdo (FEVE) de 42%. Hemograma, provas de função hepática, função renal, lactato desidrogenase e creatina kinase-MB eram normais. Foi diagnosticado edema agudo de pulmão secundário, disfunção de ventrículo esquerdo e suspensa a clozapina no primeiro dia de internação na CTI. A paciente evoluiu bem e, sete dias após a suspensão da medicação, o resultado do ecocardiograma foi normal (FEVE de 62%). Esse episódio foi diagnosticado como cardiomiopatia associada ao uso de clozapina.

Até 2002, haviam sido relatados 178 casos de cardiomiopatia associada à clozapina e cerca de 80% dos pacientes apresentavam idade inferior a 50 anos. Não houve, até o presente momento, nenhum relato desta complicação associada ao uso da clozapina no Brasil.

O presente relato de caso se propõe a alertar médicos que tratam pacientes esquizofrênicos em uso de clozapina, que devem manter um alto grau de suspeição se sinais ou sintomas de toxicidade cardíaca aparecerem. Além dos exames laboratoriais, os pacientes precisam ser instruídos a relatar qualquer sinal de miocardite (febre, cansaço, dor torácica, palpitações, taquicardia, dispnéia e edema periférico). Casos como este ajudam a reforçar a necessidade de uma avaliação eletrocardiográfica em pacientes em início de tratamento com clozapina.

Raquel De Boni
Hospital de Clínicas de Porto Alegre

Clarissa Severino Gama, Maria Inês Lobato
Serviço de Psiquiatria do Hospital de Clínicas de Porto Alegre

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