Research Paper
Article de recherche

Prevalence of tics and Tourette syndrome in an inpatient adult psychiatry setting

Valsamma Eapen, MRCPsych, PhD; Martin Laker, MRCPsych; Anita Anfield, MRCPsych; Jeremy Dobbs, MRCGP; Mary M. Robertson, MD, FRCPsych

Eapen — Faculty of Medicine and Health Sciences, U.A.E. University, Al Ain, United Arab Emirates; Laker — Southend Community Care Services, Runwell Hospital, Wickford, Essex, UK; Anfield — Longview Adolescent Unit, Colchester, Essex, UK; Dobbs — The Surgery, Cerne Abbas, Dorchester, UK; Robertson — Department of Psychiatry and Behavioural Sciences, University College London Medical School, London, UK.

Objective: Given the widely recognized genetic basis for Gilles de la Tourette syndrome (TS) and the suggestion that the putative TS gene(s) may be expressed as or associated with a variety of psychiatric illnesses, this study was undertaken to assess the prevalence of tics and TS in a psychiatric inpatient population. **Design:** Cross-sectional study. **Setting and patients:** 200 consecutive adult patients who were admitted to the psychiatric wards of University College London Teaching Hospitals. **Outcome measures:** TS and related behaviours, as assessed by the comprehensive semi-structured National Hospital Interview Schedule. **Results:** None of the 200 patients had definite TS, but 2 were observed to have motor tics; 10 had a history of tics (present for less than a year), and 7 reported a family history of tics. Thus, 19 (9.5%) inpatients qualified for inclusion in a broadly defined TS diathesis. These rates are significantly lower than those reported in a similar community based epidemiological study of adolescents ($p = 0.018$). **Conclusions:** Our findings do not support the theory that TS and related behaviours are over-represented among adult inpatients with psychiatric illnesses.

Objectif : Comme on reconnaît en général que la maladie de Gilles de la Tourette (MT) est d'origine génétique et comme on laisse entendre que le ou les gènes incriminés en théorie peuvent s'exprimer par toutes sortes de maladies psychiatriques ou y être associés, cette étude vise à évaluer la prévalence des tics et de la MT dans une population de patients hospitalisés en psychiatrie. **Conception** : Étude transversale. **Contexte et patients** : Deux cent patients adultes consécutifs admis au service de psychiatrie des University College London Teaching Hospitals. **Mesures de résultats** : MT et comportements connexes, évalués au moyen du questionnaire hospitalier national intégré (National Hospital Interview Schedule) semi-structuré. **Résultats** : Aucun des 200 patients n'avait une MT confirmée, mais on a observé que 2 avaient des tics liés à la motricité, 10 avaient des antécédents de tics (depuis moins d'un an) et 7 ont signalé des antécédents familiaux de tics. Ainsi, 19 (9,5 %) des patients hospitalisés pouvaient être inclus dans une diathèse de la MT définie de façon générale. Ces taux sont beaucoup plus bas que ceux qu'on a signalés dans le contexte d'une étude épidémiologique communautaire semblable portant sur des adolescents ($p = 0,018$). **Conclusions** : Nos constatations n'appuient pas la théorie selon laquelle la MT et les comportements connexes sont surreprésentés chez les adultes hospitalisés qui ont des maladies psychiatriques.

Correspondence to: Dr. Valsamma Eapen, Associate Professor, Faculty of Medicine and Health Sciences, United Arab Emirates University, PO Box 17666, Al Ain, United Arab Emirates; fax 971-3-7672995; veapen@uaeu.ac.ae

Medical subject headings: comorbidity; cross-sectional studies; genetic predisposition to disease; inpatients; psychopathology; tics; Tourette syndrome.

J Psychiatry Neurosci 2001;26(5):417-20.

Submitted Jul. 10, 2000
Revised Apr. 18, 2001
Accepted Jun. 7, 2001

© 2001 Canadian Medical Association

Introduction

Tourette syndrome (TS) is characterized by multiple motor and one or more vocal tics, the number, frequency and complexity of which change over time. Although the generally accepted prevalence figure is 0.5 per 1000 (approx. 110 000 patients in the United States and 25 000 in the United Kingdom), this may well be an underestimate as indicated by a prevalence of 2%–3%[1] and 0.15%–1.1%[2] in recent epidemiological studies.

It is now recognized that TS has significant genetic determinants and the mode of transmission, although slightly controversial, is thought to be by a single major locus inherited as an autosomal dominant trait or as a complex mixed model.[3] The clinical characteristics of TS patients seem independent of culture, suggesting a biologic pathogenesis.

There has recently been much interest in various psychopathologic disorders and behaviours associated with TS, including obsessive–compulsive disorder (OCD), attention deficit hyperactivity disorder (ADHD), depression and anxiety disorders. There is, however, some disagreement about the nature of the relation between these conditions and TS. For example, although OCD is widely recognized as both clinically and etiologically related to TS,[4] there is some debate about the link between ADHD and TS. Some suggest that the increased occurrence of ADHD among patients with TS represents a different manifestation of the TS diathesis,[5] whereas others suggest that the 2 disorders may be etiologically related in only some individuals with TS[6] or that this co-occurrence may result from an overlap at biochemical, neuroanatomic or symptomatic levels.[7] A variety of childhood behavioural disorders including conduct and oppositional defiant disorder have been suggested to be genetically related to TS.[5] Through a mechanism involving the additive and subtractive effect of 3 dopaminergic genes, it has also been proposed that many disruptive behavioural disorders (e.g., ADHD, TS, stuttering, learning disabilities, substance abuse, conduct and oppositional defiant disorders) are inter-related, have a strong genetic component and may be polygenically inherited.[8] In fact, Comings[5] and colleagues[8] suggests that the putative TS gene(s) is a common gene(s) that may be associated with many other psychiatric disorders and may be the common thread between these disorders and TS. He compared the frequency of occurrence of psychiatric problems in non-proband relatives with TS and relatives without TS and concluded that TS is a behavioural spectrum disorder and that there is evidence of a genetic association between TS and, for example, learning disorders, dyslexia, stuttering, autism, conduct disorder, depression, mood swings, mania, irritability, short temper, anxiety with panic attacks, phobias, speech problems, inappropriate sexual behaviours and addictive behaviours.[5,9] However, this theory remains controversial; other studies have failed to find such associations.[3,6]

Patients with TS have been reported to have higher rates of some psychiatric conditions such as anxiety and depression than either controls or the general population.[3] Shapiro et al,[10] on the other hand, failed to find an association between TS and any specific psychiatric syndrome or psychodynamic factors. In a controlled study using the Minnesota Multiphasic Personality Inventory, a group of patients with TS did not differ significantly from general outpatients on factors such as overt and underlying psychosis, obsessive–compulsive traits, inhibition of hostility, hysteria and general maladjustment. However, only 1 patient of 36 was free from psychiatric illness, most being diagnosed as having various types of "personality disorders." This is in keeping with the findings from a recent controlled study reporting higher rates of personality disorder depression, anxiety and obsessionality among patients with TS.[11] Kerbeshian et al,[12] who found a higher than expected rate of bipolar disorder and major depression in a sample of patients with TS, offer an explanatory model involving the canalization of basal-ganglia-mediated dysfunctions,[12] whereas others suggest the involvement of a number of common genes that affect dopamine and serotonin may be responsible.[9]

If there is a common TS gene(s) and other psychiatric conditions are also associated with the TS gene(s), one would expect to find higher rates of TS and related behaviours among patients in psychiatric hospitals. Although many previous investigations have focused on patients with TS and their families, to the best of our knowledge, none have assessed TS prevalence rates in a psychiatric population. We therefore examined the prevalence of tics and related behaviours in an adult population in various psychiatric wards.

Methods

Two hundred consecutive admissions to general adult psychiatric wards of University College London Teaching Hospitals (Camden and Islington Community

Health Services NHS Trust) were interviewed using the National Hospital Interview Schedule (NHIS) for the assessment of TS and related behaviours. This semistructured interview schedule has been widely used and the psychometric properties have been established.[13] Information was gathered regarding the presence or absence of TS, tics and related behaviours; the interview schedule is designed to determine both tics at present and tics ever (life time). Data were corroborated with hospital notes and information obtained from family members. Individuals were assigned to diagnostic categories as follows: (1) definite TS or tics (i.e., satisfying *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition, revised [DSM-III-R], criteria on history and examination), (2) possible TS or tics (i.e., symptoms on history or examination, but not both). Because this was an adult sample and the patients were not admitted for tic-related problems, the age criteria (onset before 21 years of age), as per the DSM-III-R, was not applied before a diagnosis was made.

Results

No patient satisfied the criteria for TS by having had both motor and vocal tics, concurrently or otherwise, on and off for a period of 1 year or more. Two patients (1%) demonstrated motor tics at interview, but these patients did not indicate a history of tics, and no vocal tics were observed. A further 10 (5%) patients gave a history of tics, which were either motor or vocal, but not both, and were reported to have been present for less than a year. None of these patients indicated a positive family history. However, 7 (3.5%) other patients reported a family history of tics, but none gave a family history of TS. The psychiatric diagnoses of the inpatients, as per the clinical modification of the *International Classification of Diseases, 9th revision*, were as follows: depression, $n = 62$; schizophrenia, $n = 45$; alcohol abuse, $n = 26$; mania, $n = 25$; personality disorders, $n = 15$; drug abuse, $n = 10$; drug-induced psychoses, $n = 9$; paranoid psychosis, $n = 8$; OCD, $n = 7$; adjustment reaction, $n = 6$; mixed affective disorder, $n = 5$; phobias, $n = 4$; anorexia nervosa, $n = 2$; anxiety disorder and panic attacks, $n = 2$; bulimia nervosa, $n = 1$; chronic organic brain syndrome, $n = 1$; other diagnoses, $n = 9$.

Discussion

Twelve of 200 (6%) patients in this study were classified as having "possible tics" (motor or vocal tics ever, by history or examination). This is significantly lower than the 18% reported by Mason et al[1] in an epidemiological study of a mainstream secondary school population using the same instruments ($p < 0.001$). There have been suggestions that the prevalence of TS is 10 times greater in children and adolescents than in adults[14] and that 50% of patients with TS lose most of their tics by the age of 18 years.[15] We therefore considered that our rates were low because our study was performed in an adult population; we do not think that this occurred, however, because the NHIS has questions about tics ever, and this would include the childhood years. Even if we were to consider the actual rate of tics to be 6% (versus 18% in 13–14 year olds[1]), our results suggest that the prevalence of tics is not higher among patients with psychiatric illnesses. With regard to the 2 patients observed to have motor tics at interview, it is difficult to confirm if they had TS. If we were to assume that they may have had vocal tics and would thus be categorized as having "possible TS" (motor and vocal tics diagnosed by either history or by examination but not both), our TS prevalence would be 1% — still lower, but not significantly lower, than the 3% rate observed for "definite TS" by Mason et al.[1]

There are reports that patients seeking treatment for psychiatric problems have been noted, for the first time and as an incidental observation, to have TS diathesis.[16,17] We observed a prevalence of 9.5% for a broadly defined TS diathesis which had not been previously observed in these patients. Thus, although clinicians should be vigilant as to the possibility of tics and related behaviours in patients seeking psychiatric help, the prevalence of these behaviours does not seem to be higher than in the general population. Furthermore, the fact that our patients had either motor or vocal tics (but not both) and they had been present only for less than 1 year makes it unlikely that our patients had TS. It has been suggested that the frequency of occurrence of such transient and chronic motor tics are much higher than TS in the general population and in TS families, and that at least in some individuals, they may not be etiologically or genetically related to TS.[18]

There is now strong evidence from epidemiologic, clinical, phenomenological, biochemical and genetic studies that behaviours such as OCD may be an integral part of TS.[3,4] As well, ADHD may be genetically related to TS in some individuals, but a comorbid condition in others.[6,7] Other psychiatric conditions found to co-occur with TS include depression, anxiety and per-

sonality disorder.[11] The frequently observed comorbidity between personality disorder, depression and anxiety may partly explain this finding,[19] or the higher rates of anxiety and depression in patients with TS may be due to the chronic and debilitating nature of the disorder. In a controlled study, the only one to date to control for depression, Robertson[3] found that patients with TS are disproportionately obsessional and this cannot be accounted for by depression. It should be considered that the high rates reported for psychopathologic conditions in TS clinic populations may well reflect referral biases because studies on mild community-based individuals with TS do not report an increase in psychopathology apart from obsessional behaviour.[3,7]

The high prevalence of psychopathologic conditions among TS subjects may reflect comorbidity in only a subpopulation of TS patients.[20] This suggestion was based on the observation that bipolar disorder was more common in TS patients with mild tic symptoms and was invariably associated with a high lifetime prevalence of general psychopathology (e.g., generalized anxiety disorder, OCD, panic, phobias, eating disorders, self injurious behaviours, ADHD, impulse control disorders and personality disorders). Another theory that may explain higher rates of psychiatric conditions among patients with TS is that a number of common genes that affect dopamine and serotonin metabolism are involved in TS and that because these neurotransmitters modulate the function of many areas of the brain, this results in a wide spectrum of behavioural disorders.[9] However, if this is the case, one would expect the reverse to be true as well — that is, a higher rate of TS among patients with psychiatric illnesses. Our results suggest that even a broadly defined TS diathesis is not over-represented among psychiatric patients. However, our study is limited because of the relatively small sample size and the lack of collateral histories.

Conclusion

Our findings do not support the suggestion that TS is over-represented among the adult inpatient psychiatric population, and that TS may be associated with a wide variety of disorders, the severity of which necessitates admission to a psychiatric unit. Higher rates of comorbid conditions reported in some studies may well be due to referral biases. To determine if TS is associated with other psychopathologic disorders, future studies should include nonreferred mild cases of TS in the community.

Competing interests: None declared.

References

1. Mason A, Banerjee S, Eapen V, Zeitlin H, Robertson MM. The prevalence of Tourette syndrome in a mainstream school population. *Dev Med Child Neurol* 1998;40:292-6.
2. Kadesjo B, Gillberg C. Tourette's disorder: epidemiology and comorbidity in primary school children. *J Am Acad Child Adolesc Psychiatry* 2000;39(5):548-55.
3. Robertson MM. Tourette syndrome, associated conditions and the complexities of treatment. *Brain* 2000;123(3):425-62.
4. Eapen V, Robertson MM, Alsobrook JP, Pauls DL 2nd. Obsessive compulsive symptoms in Gilles de la Tourette syndrome and obsessive compulsive disorder: differences by diagnosis and family history. *Am J Med Genet* 1997;74:432-8.
5. Comings DE. *Tourette syndrome and human behaviour*. California: Hope Press; 1990.
6. Pauls DL, Leckman JF, Cohen DJ. Familial relationship between Gilles de la Tourette syndrome, attention deficit hyperactivity disorder, learning disabilities, speech disorders and stuttering. *J Am Acad Child Adolesc Psychiatry* 1993;32:1044-50.
7. Eapen V, Robertson MM. Gilles de la Tourette syndrome and attention deficit hyperactivity: no evidence for genetic relationship. *Neuropsychiatry Neuropsychol Behav Neurol* 1996;9:192-6.
8. Comings DE, Chiu C, Ring RH, Gade R, Ahn C, MacMurray JP, et al. Polygenic inheritance of Tourette syndrome, stuttering, attention deficit hyperactivity, conduct and oppositional defiant disorder: the additive and subtractive effect of the three dopaminergic genes — DRD2, D beta H and DAT1. *Am J Med Genet* 1996;67:264-88.
9. Comings DE. Tourette syndrome, a behavioural spectrum disorder. *Adv Neurol* 1995;65:293-303.
10. Shapiro AK, Shapiro E, Bruun RD, Sweet RD. *Gilles de la Tourette syndrome*. New York: Raven Press; 1978.
11. Robertson MM, Banerjee S, Fox-Hiley PJ, Tannock C. Personality disorder and psychopathology in Tourette's syndrome: a controlled study. *Br J Psychiatry* 1997;171:283-6.
12. Kerbeshian J, Burd L, Klug MG. Comorbid Tourette's disorder and bipolar disorder: an aetiologic perspective. *Am J Psychiatry* 1995;152:1646-51.
13. Robertson MM, Eapen V. The National Hospital Interview Schedule for the assessment of Gilles de la Tourette syndrome and related behaviours. *Int J Meth Psych Res* 1996;6:203-26.
14. Burd L, Kerbeshian J, Wikenheiser M, Fisher W. Prevalence of Gilles de la Tourette syndrome in North Dakota adults. *Am J Psychiatry* 1986;143:787-8.
15. Leckman JF, Zhang H, Vitale A, Lahnin F, Lynch K, Bondi C, et al. Course of tic severity in Tourette syndrome: the first two decades. *Paediatrics* 1998;102:14-9.
16. Sverd J. Clinical presentations of the Tourette syndrome diathesis. *J Multihand Pers* 1989;2:311-27.
17. Minderaa RB, van Gemert TM, van de Wetering BJM. Onverwachte presentanewijzen van het syndroom van Gilles de la Tourette. *Tijdschrift voor Psychiatrie* 1988;30:246-54.
18. Eapen V, Robertson MM. All that tics may not be Tourette's [letter]. *Br J Psychiatry* 1994;164:708.
19. Flick SN, Roy-Byrne PP, Cowley DS, Shores MM, Dunner DL. DSM-III-R personality disorders in a mood and anxiety disorders clinic: prevalence, comorbidity, and clinical correlates. *J Affect Disord* 1993;27:71-9.
20. Berthier ML, Kulisevsky J, Campos VM. Bipolar disorder in adult patients with Tourette syndrome: a clinical study. *Biol Psychiatry* 1998;43:364-70.