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Gilles de la Tourette syndrome – a case report from Guyana in South America

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A case of the Gilles de la Tourette syndrome from Guyana in South America is presented. The patient had a positive family history as well as coprolalia, echolalia, and attention deficit disorder with hyperactivity. The family history and cross-cultural similarity emphasise the biological factors in the aetiology of the syndrome.

INTRODUCTION

The Gilles de la Tourette syndrome (GTS) was once thought to be a bizarre rarity, but is now recognised as being more common than was previously thought (Robertson, 1989). In addition, many cases with mild symptoms do not come to medical attention (Caine *et al.*, 1988; Kurlan *et al.*, 1986; Robertson and Gourdie, 1990). GTS is diagnosed by the presence of both motor and vocal tics; however, associated features such as echophenomena (copying behaviours), coprophenomena (inappropriate uttering of obscenities or making of obscene gestures), and obsessional thoughts and behaviours are also recognised as being important parts of the syndrome (Robertson *et al.*, 1988; Robertson, 1989). GTS is found in all cultures and racial groups, and substantial cohorts of GTS patients have been reported from North America, the United Kingdom and Europe, but from most other parts of the world, to date, only case reports exist (Robertson, 1989). To the best of our knowledge no cases of GTS have been reported in the Anglo Saxon literature from South America.

A GTS from Guyana in South America is reported and we emphasise not only the cross-cultural similarities of GTS patients, in that our patient is not markedly different from those documented previously, but also note the positive family history, which is in accord with the notion that GTS has a biological and genetic basis.

CASE REPORT

A 45-year-old man from Guyana was admitted to our hospital following a serious suicidal attempt by carbon monoxide poisoning. Over the 2-3 days prior to admission, he had suffered major financial losses which prompted the suicidal attempt. After admission, on the ward, he was noticed to have several motor and vocal tics and further detailed evaluation confirmed a diagnosis of GTS.

His GTS symptoms had begun at the age of 13, although the diagnosis of GTS had never been made. Indeed, his symptoms were so mild that he did not consider them a problem, and had thus never sought medical help for them. We examined him using a semi-structured interview previously used to diagnose and assess GTS phenomenology (Robertson *et al.*, 1988; Robertson *et al.*, 1989; Robertson and Gourdie, 1990).

It transpired that at the age of 13, he developed eye blinking which progressed over the next 2 years to include eyebrow raising, a nasal twitch, platysma tightening, shoulder shrugging, drumming of the fingers, flexion and extension movements of the leg, swallowing and abdominal contractions. He had several vocal tics such as sniffing, throat clearing and a "nervous" cough. However, it is interesting to note that coprolalia (inappropriate involuntary uttering of obscenities) started only when he was 36 years of age. He was also noted to have echolalia, and, as a child, admitted to having had the symptoms of attention deficit disorder with hyperactivity. His GTS symptoms showed a waxing and waning course, and he could suppress them voluntarily for several minutes at the expense of inner tension. He had noticed the tics to be worse when he was under stress and improved while he read.

Our patient was born in Guyana after a normal pregnancy, but he possibly suffered a perinatal birth injury, in that he was delivered by forceps after a prolonged and difficult labour. He was of mixed ethnic origin, but judging by his slanted/mongoloid eyes and one of his names, there was almost certainly some oriental influence. He had no significant childood illnesses or traumas. He came to Britain as a young boy, aged 12, and completed his senior

Cambridge certificate at the age of 16. After leaving school he maintained a fairly good occupational record and had functioned normally in his social life. At one stage aged 22 years he had a period of impulsive gambling, self-injurious behaviour (a single episode of wrist-slashing), and at the same time he was also prone occasionally to aggressive outbursts.

His past psychiatric history included a brief period of depression some 18 months previously, when he responded well to dothiepin at a dose of 225 mg/day, which he continued for 3 months. The family history was noteworthy in that his brother, aged 43, apparently had facial tics, a nasal twitch and excessive sniffing (a vocal tic) which had started during his early teens, thus being a probable GTS case (diagnosed on history only—see Robertson and Gourdie, 1990).

Mental state examination revealed an alert, cooperative intelligent man with multiple motor and vocal tics and a depressed mood and affect with suicidal ideation. He showed no evidence of psychosis nor cognitive impairment. Physical examination was unremarkable and special investigations were within normal limits. This included normal full blood count with no acanthocytes, urea and electrolytes, serum copper (11.7 mm/l) and caeruloplasmin (0.45 m/l).

A diagnosis of depression in the setting of GTS was made, and the patient was prescribed paroxetine 20 mg. As his GTS symptoms were mild, he was not offered a dopamine antagonist. He was discharged when thought to be no longer a suicidal risk and was given an out-patient appointment for follow-up.

DISCUSSION

We present the first case of GTS from South America in the Anglo Saxon literature, demonstrating that it does occur on that continent. What is perhaps more important is that the phenomenology of the GTS is similar to that documented world-wide, with both motor and vocal tics, coprolalia, echolalia, aggression and attention deficit disorder with hyperactivity; in addition there is a positive family history of GTS (Robertson, 1989). The phenomenological and genetic cross-cultural similarity suggest a biological rather than a psychosocial aetiology to the disorder.

It is interesting that our patient's coprolalia only began at the relatively late age of 36, as it usually has it's onset around 13–14.5 years of age (Robertson, 1989). Coprophenomena and obsessionality have been shown to be related (Robertson *et al.*, 1988) and it is interesting therefore that our patient showed no obsessionality

Our patient also had two depressive episodes, and in this context it has been shown that GTS patients become depressed and that the depression is related to increasing age and the presence of echophenomena (Robertson *et al.*, 1988). Our patient was 44 years old when he first become depressed and he does have echolalia, both of which would be in agreement with these previous findings. Suicide has not to date been reported in GTS, but such cases are known (Robertson and van de Wetering, in preparation).

Our patient's gambling appeared to be of an impulsive nature in that he would have sprees of gambling, and be relatively normal in between times. His aggression, similarly, occurred in bouts. Aggression is well documented in patients with GTS (Robertson, 1989), while an association with gambling has also been reported (Comings, 1990). Both may be seen as disorders of inhibition and/or impulse control, which has been suggested as the basis to GTS (Comings, 1990).

GTS is thought to be genetic and may manifest as GTS or chronic multiple motor or vocal tics only (Kurlan *et al.*, 1986; Pauls *et al.*, 1981; Robertson and Gourdie, 1990) or obsessional behaviours (Comings and Comings, 1987; Pauls and Leckman, 1986; Pauls *et al.*, 1986a, b). Our patient had a positive family history of probable GTS, which is in agreement with this notion.

Our patient with GTS from Guyana in South America supports the argument for GTS being primarily a biologic condition with a genetic basis.

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