Correspondence

EDITED BY LOUISE HOWARD

Rabbit syndrome treated with olanzapine

We wish to share with you our experience in treating a person with schizophrenia who developed rabbit syndrome. Rabbit syndrome, characterised by rapid, rhythmic orofacial movements, often accompanied by lip sounds (Schwartz *et al*, 1995) is considered one of four tardive dyskinesia (TD) variants, all of which are extrapyramidal side-effects of long-term neuroleptic treatment (Inada *et al*, 1991). The incidence of rabbit syndrome among TD variants is about 2.4% (Wada & Yamaguchi, 1992), with a mean prevalence of 15–20% among neuroleptic-treated patients in general (Baldessarini, 1988) and 9.9% in psychiatric hospital populations. Olanzapine is hereby suggested as a possible solution for alleviating rabbit syndrome without risking the emergence of extrapyramidal side-effects and simultaneously combating schizophrenic symptoms.

A 28-year-old single woman, suffering from schizophrenia for nine years, had been treated by depot injections of zuclopenthixol (200 mg every fortnight) and with biperiden (4 mg/day) since 1995. Her mental state deteriorated and she was hospitalised in an acute psychotic state, suffering from paranoid delusions. Signs of typical rabbit syndrome were evident.

In seeking out an appropriate treatment strategy for her psychotic state and the rabbit syndrome (which did not seem to respond to biperiden), it was decided to stop administration of zuclopenthixol and to initiate olanzapine, starting at 5 mg/day and reaching maximal dosage (10 mg/day). Within 2–3 weeks, not only had her psychotic symptoms disappeared, but improvement was also observed in her rabbit syndrome. Her Abnormal Involuntary Movement Scale (AIMS, Wojcik *et al*, 1980) score dropped from 14 to 8. After 25 days of hospitalisation, she was released into community care and continued to receive olanzapine (10 mg/day). Follow-up, a year later, found her to be in remission from the psychosis, with no signs of rabbit syndrome, under olanzapine treatment.

It has been postulated that the underlying mechanism of rabbit syndrome is supersensitivity of dopamine receptors, possibly due to an underlying predisposition. It has also been suggested that rabbit syndrome is a result of multiple system atrophy (Nishiyama *et al*, 1993). The most prominent, albeit controversial, treatment agents for rabbit syndrome are benzhexol and biperiden. We were anxious to avoid aggravation or uncovering of TD by the addition of anticholinergic agents. Olanzapine displays high affinity for type 2 serotonin ($5\text{-}HT_2$) receptors and, although the activity ratio between $5\text{-}HT_2$ and type 2 dopamine (D_2) receptors is slightly lower than for clozapine, it is still about twice as active at $5\text{-}HT_2$ than at D_2 receptors. Hence, olanzapine is less likely to be associated with TD, and its application in preventing or ameliorating rabbit syndrome seemed appropriate (O'Brien & Barber, 1998).

This case demonstrates the possible usefulness of olanzapine as a mono-drug treatment strategy for dealing with rabbit syndrome triggered by typical neuroleptics and simultaneously treating psychotic symptoms.

Baldessarini, R. J. (1988) A summary of current knowledge of tardive dyskinesia. *Encéphale*, **14**, 263–268.

Inada, T., Yagi, G., Kaijima, K., *et al* (1991) Clinical variants of tardive dyskinesia in Japan. *Japanese Journal of Psychiatry and Neurology*, **45**, 67–71.

Nishiyama, K., Masuda, N. & Kurisaki, H. (1993) A case of rabbit syndrome – its unique pharmacological features. *Rinsho Shinkeigaku – Clinical Neurology*, **33**, 663–665.

O'Brien, J. & Barber, R. (1998) Marked improvement in tardive dyskinesia following treatment with olanzapine in an elderly subject (letter). *British Journal of Psychiatry*, **172**, 186.

Schwartz, M., Weller, B., Erdreich, M., *et al* (1995) Rabbit syndrome and tardive dyskinesia: two complications of chronic neuroleptic treatment. *Journal of Clinical Psychiatry*, **56**, 212.

Wada, Y. & Yamaguchi, N. (1992) The rabbit syndrome and antiparkinsonian medication in schizophrenic patients. *Neuropsychobiology*, **25**, 149–152.

Wojcik, J. D., Gelenberg, A. J. & Labrie, R. A., *et al* (1980) Prevalence of tardive dyskinesia in an outpatient population. *Comprehensive Psychiatry*, **21**, 370–380.

R Durst, G. Katz, J. Zislin, S. Raskin, I. Kalman The Jerusalem Mental Health Center, Kfar Shaul Hospital, affiliated with the Hebrew University, Hadassah Medical School, Jerusalem, 91060 Israel

Serotonin transporters in ecstasy users

Semple *et al* (1999) report a reduction *in vivo* of ^{123}I-labelled 2β-carbomethoxy-3β-(4-iodophenyl)tropane (β-CIT) uptake in the cerebral cortex of 3,4-methylenedioxymethamphetamine (MDMA, 'ecstasy') users. They interpret this observation to be an indication of a decrease in serotonin transporters in the cortex of MDMA users. However, there are serious methodological concerns with this interpretation of their data.

It has been demonstrated that the radioligand [^{123}I]β-CIT binds with high affinity to dopamine, serotonin and noradrenaline transporters in human brain (Farde *et al*, 1994; Laruelle *et al*, 1994). In the case of the serotonin transporter, *in vivo* displacement of β-CIT binding by selective serotonin reuptake inhibitors (SSRIs) has established that specific (displaceable) binding occurs in brainstem and thalamus (Laruelle *et al*, 1993; Pirker *et al*, 1995; Tauscher *et al*, 1999). However, this is not true for the cerebral cortex. Indeed, Laruelle *et al* (1993) observed that [^{123}I]β-CIT uptake in cortical areas was unaffected by citalopram administration in non-human primates. Similarly, recent SSRI displacement studies of [^{11}C]McN-5652, a selective serotonin transporter radioligand for positron emission tomography (PET) imaging, failed to observe specific binding in the cerebral cortex (Parsey *et al*, 1999). The lack of evidence for specific binding to serotonin transporters in the cerebral cortex *in vivo* is not surprising when one considers the paucity of these transporters in primate cortex (Jagust *et al*, 1996). We are aware of only a single report of apparent displacement of β-CIT by citalopram in primate cortex:

https://doi.org/10.1192/bjp.176.2.193 Published online by Cambridge University Press