Letter to the Editor

Ziprasidone Might Prevent Life-Threatening Hypo- and Hyperthermia Induced by Antipsychotics

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The report presented here adds to the very sparse literature reporting temperature deviations with antipsychotics. The case suggests that high-dose treatment with the second-generation antipsychotic ziprasidone may prevent recurrent dysregulation of body temperature during long-term psychopharmacotherapy. Hyperthermia is defined as drop in core body temperature below 35°C (95°F). Hypothermia as well as drug fever or hyperthermia are known as rare, serious and unpredictable side effects, so-called type B reactions, of both typical and atypical antipsychotic agents (Van Marum et al., 2007).

A 52-years old caucasian woman with a body mass index of 28 and without family, perinatal as well as substance abuse history suffers from chronic disorganized schizophrenia (DSM-IV: 295.10; ICD-10: F20.1). She displays disorganized thinking, grossly disorganized and aggressive behaviour as well as absent or inappropriate emotional expression over two decades. The dysregulations of body temperature occurred preferentially following start or dose increase of an antipsychotic and lasted the last five years with nearly four monophasic events per year. She consecutively evolved hypothermias down to 32.0°C rectal and hypothermia-accompanied bradycardias down to 32/min. She also developed hyperthermias up to 40.0°C rectal, additionally repeated subclinical hypoglycaemias and somnolence to coma. During these periods she was treated with benperidol combined with levomepromazine or melperone, pipamperone with and without amisulpride, promethazine as well as zuclopenthixole (Löffler et al., 2007). Rather moderate dysregulations of temperature were observed during therapy nearly with all other typical and atypical antipsychotics except clozapine, ziprasidone, aripiprazole and paliperidone, which so far she did not receive. Body temperature normalized after antipsychotic drug discontinuation always within hours. Detailed screenings in internal medicine, toxicology, neurology as well as in neuropsychology did not reveal any pathbreaking finding.

Ziprasidone was chosen because of its relatively weak D2 receptor antagonism combined with a 5-HT1A receptor agonism which is quite different from the majority of other typical and atypical antipsychotics. This view is supported by the WHO database showing a low incidence of hypothermia for ziprasidone (Van Marum et al., 2007). High-dose therapy was necessary to improve severe excitements and to avoid antipsychotic co-medication, respectively. Now under ziprasidone 320 mg/day (off-label use), lorazepam 8 mg/day and pantoprazole 40 mg/day her care home ability returned and her body temperature varied physiologically at least over one year (prior to ziprasidone 32.0-40.0°C rectal, following ziprasidone 36.0-37.5°C rectal).

In conclusion, (i) ziprasidone might be useful in treating patients with dysregulation of body temperature under antipsychotics (ii) ziprasidone long-term therapy with 320 mg/day (off-label use) was well tolerated and (iii) in persons who cannot control their physical status, core body temperature should be supervised closely, especially when a long-term medication with antipsychotic drugs is changed.
References
