Molecular Genetics and Metabolism Reports 13 (2017) 95–96

Contents lists available at ScienceDirect

Molecular Genetics and Metabolism Reports

journal homepage: www.elsevier.com/locate/ymgmr

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Weight loss induced by quetiapine in a 22q11.2DS patient

The 22q11.2 deletion syndrome (22q11.2DS) is the most frequent known genetic risk factors for schizophrenia [1]. Here, we present an unusual weight loss in a 22q11.2DS patient given quetiapine.

The patient was a 37-year-old man (5-ft 7-in. tall; 145,2 lb) with a 22q11.2DS. At the age of 20, he developed a schizophrenia and failed to benefit from treatments by atypical antipsychotics and haloperidol. Weight always remained stable.

After 3 weeks of antipsychotics withdrawal, a treatment by quetiapine was progressively introduced (up to 300 mg/day). Twelve weeks later, an anorexia with worrisome weight loss (− 17,6 lb) was noted. Results of the metabolic work-up (chemistry screening, leptin and ghrelin levels, CBC, CPK, thyroid function tests, oral glucose tolerance test and abdominal echography) were unremarkable. The quetiapine was discontinued and the patient recovered his initial weight two months later (145 lb). Resumption of quetiapine was proposed. As a result, an anorexia with weight loss recurred and the treatment was interrupted.

Quetiapine is a second-generation antipsychotic that has affinity for D2, 5-HT2A, H1, alpha 1 and 5-HT1A receptors but its mechanism of action remains unknown. In particular, quetiapine has been shown to occupy approximately 49.1% of D2 receptors at therapeutic doses [2,3], which is lower than other antipsychotics (olanzapine: 96.5%, aripiprazole: 86.9%, risperidone: 92.4%, haloperidol: 91.9% and clozapine: 61.7%). Among the 22q11.2 genes, catechol-*O*-methyl-transferase haploinsufficiency decreases the degradation of dopamine, causing higher levels of catecholamines in the CNS. COMT-dependent degradation is of importance in brain regions with low expression of the presynaptic transporter, such as the prefrontal cortex [4]. This dopaminergic-rich region has been shown to be important to food-related decision-making [5]. Therefore, COMT haploinsufficiency, in addition with the atypical pharmacological profile of quetiapine, gives insights to understand appetite reduction in this case report. In conclusion, taking into account molecular diagnosis in psychiatry promises to improve patient care though advances in personalized medicine.

Ethics approval and consent to participate

Written informed institutional consent was obtained from the patient for the publication of personal details in this manuscript. The consent form is kept with the patient's clinical notes and available for review by the Editor-in-Chief.

Consent to participate

The patient gave his consent to participate.

Availability of data and supporting materials section

Data are available by e-mail to caroline.demily@ch-le-vinatier.fr

Competing interests

None.

Funding

None.

Authors' contributions

CD collected the data. CD, AP, FT, NF wrote the paper. All authors approved the final version of the manuscript.

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Received 29 September 2017; Accepted 1 October 2017
Available online 17 October 2017

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