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Rhabdomyolysis secondary to quetiapine

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Quetiapine is one of the drugs used most in current Psychiatry due to its therapeutic efficiency and clinical safety. We report the case of a 26-year-old male patient with severe mental retardation due to Y-chromosome partial deletion who initiated treatment with quetiapine for the control of his aggressiveness, and who developed severe rhabdomyolysis two weeks later. In spite of the confirmed clinical safety of quetiapine, doctors must monitor the appearance of rare but serious adverse effects as that presented in this clinical case.

Keywords:
Quetiapine, Rhabdomyolysis, Mental retardation, Drug toxicity

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Rabdomiólisis secundaria a tratamiento con quetiapina

La quetiapina es uno de los fármacos más empleados en la Psiquiatría actual, debido a su eficacia terapéutica y seguridad clínica. Presentamos el caso de un paciente de 26 años, con retraso mental grave debido a deleción parcial del cromosoma Y, que inicia tratamiento con quetiapina para el control de su agresividad, y que desarrolla a las 2 semanas una rabdomiólisis severa. A pesar de la seguridad clínica contrastada de la quetiapina, los médicos deben vigilar la aparición de raros pero graves efectos adversos como el presentado en este caso clínico.

Palabras clave:
Quetiapina, Rabdomiólisis, Retraso mental, Toxicidad medicamentosa

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Quetiapine is one of the antipsychotic drugs used most in the treatment of behavioral disorders. According to the data sheet approved in our country, its indications include treatment of schizophrenia, moderate or severe manic episodes and major depressive episodes in bipolar disorder (not the prevention of recurrence of manic or depressive episodes).[1] Among its most frequent adverse effects (>10% of the patients) are those that affect the CNS (dizziness, somnolence and headache), alterations (dyslipidemia) and gastronintestinal (xerostomy and weight gain) metabolic, although different alterations may be produced with much less frequency: leukopenia, increase in transaminasas, blurred vision and others.

Elevations of creatine kinase (CK) have been described previously in patients with schizophrenia, mania and psychotic depression. In these patients, the elevations of CK during the acute psychotic episode are not necessarily related with an elevated physical activity during the episodes but are a manifestation of a variety of neuromuscular dysfunctions characteristic of psychotic patients (especially those with schizophrenia).[2] Other causes of elevation of CK in psychiatric patients are intake of drugs, seizures, traumatisms and different infectious diseases.

We present the case of a 26-year old male with moderate mental retardation (Y chromosome partial deletion), frequent episodes of self- and hetero-aggressiveness, who received treatment with risperidone (1 mg /12 hours), topiramate (50 mg / 8 hours), oral rivotril solution (6.5 mg / 24 hours, divided into 4 doses), flurazepam (30 mg/24 hours), biperidene retard (4 mg/24 hours) and due to the refractariety of his aggressiveness, with oral quetiapine since 2 weeks ago (200 mg). One week prior to his hospital admission, he began to have more irritability than usual, refusal to obey orders from his caregivers and symptoms of poorly defined abdominal pain without other respiratory, digestive or neurological symptoms of importance. In the physical examination, no relevant data were found except for superficial lesions that the patient himself had inflected (bites on his hands). In the lab work

requested in the Emergency Service, the following results were seen: Complete blood count: 7,100 leukocytes, with normal formula; hemoglobin 13.1 g/dL, platelets 17,4000 / mm^3; Biochemistry: glucose 107 mg/dL, creatinine 0.8 mg/dL; GOT 312 IU/L; GPT 185 IU/L; CK 4267 mg/dL (normal range of the laboratory until 200), mass CK-MB 117.9 IU/L (normal until 7.2), Sodium 142 mEq/L, Potassium 3.2 mEq/L and Calcium 9.6 mg/dl. No alterations were observed in the urine. Blood gas values were normal and ECG had sinus bradycardia at 42 beats per minutes.

With the diagnosis of rhabdomyolysis, it was decided to admit him to the hospital and initiate intravenous treatment with glucose-saline solution, observing progressive decrease of the CK in the following controls. In a previous admission due to behavioral alterations, serological tests for hepatitis B, C and HIV were made (all with negative result). Due to the absence of any infection, laboratory test abnormality, alcohol or drug abuse, traumatisms or seizures, it was considered that the CK was very probably due to quetiapine-induced rhabdomyolysis. Therefore its administration was discontinued. In accordance with Karch and Lasagna,[3] we should consider the relation between quetiapine intake and the adverse event observed (rhabdomyolysis) as "likely" because the reaction followed a reasonable time sequence after the administration of the medication and because this relation was confirmed on its withdrawal. However, because it is not possible to rule out a drug interaction with the other drugs the patient was taking, this causal relation should be considered as "possible" in accordance with other algorithms that evaluate the adverse reactions to drugs.[4]

Quetiapine-induced rhabdomyolysis has only been described very sporadically,[5] and has never been described in our country according to the bibliographic search made. Given the large amount of patients who receive treatment with this drug and its relatively short time on the market, we consider that this possible adverse effect must be monitored.

DECLARATION OF CONFLICT OF INTEREST

All the authors declare they have no conflict of interest to declare

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