

CASE REPORT

Rhabdomyolysis with Quetiapine

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Abstract

Although the incidence of severe side effects like neuroleptic-malignant-syndrome or rhabdomyolysis seems to be considerably lower in second generation antipsychotics compared to first generation compounds, the increased use of these substances is accompanied by patients suffering from these unwanted side effects. It is of importance to be aware of the clinical presentation of such cases and to act quickly to avoid lasting sequelae for our patients. Here we report a case of rhabdomyolysis in a male patient treated with quetiapine because of schizoaffective disorder with manic and psychotic symptoms who presented with muscle stiffness to the emergency room of our hospital. After immediate discontinuation of quetiapine the clinical symptoms and laboratory changes remitted completely within a few day (German J Psychiatry 2008; 11: 79-80).

Keywords: Quetiapine, rhabdomyolysis, neuroleptic-malignant-syndrome

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Some case reports suggest that quetiapine may induce neuroleptic-malignant-syndrome (Singh et al.; 2002, Stanley and Hunter; 2000, Whalley et al.; 1999). Since the second generation antipsychotics are increasingly prescribed and commonly regarded as being more benign in their side effect profile, attention should relentlessly be redirected to serious adverse events.

Mr. W., a 37 year old Caucasian diagnosed with schizoaffective disorder with manic and psychotic symptoms about 3 months ago, received quetiapine 500mg/die and valproic acid 1000mg/day for 10 weeks. He enjoyed a near complete remission of his psychopathological symptoms. His medical records did not include comorbidities and he had never received antipsychotics before. An epileptic fit or an infection, as well as greater physical activity or sports or alcohol, could be ruled out to cause the following symptoms:

Mr W. presented with severe muscle pain and stiffness suddenly starting in his neck on the morning of the admission date subsequently spreading from the upper to the lower

extremities in the afternoon. He reported hyperhidrosis for the previous night and showed tremor of the hands. We found muscle rigidity in the lower extremities. The muscles felt tense and warm on inspection. There were signs of a slight muscle weakness. Body temperature at admission was subfebrile at 37.9 °C, later the temperature normalized fully. Blood pressure was 90/60 mmHg, pulse 88/min. The physical examination was unremarkable otherwise. The patient was puzzled by the unfamiliar symptoms and felt sick. Laboratory results revealed a beginning rhabdomyolysis: Creatine phosphokinase 766 U/l, myoglobine 216 ng/ml, aspartate aminotransferase 30 U/l. The values of potassium, creatinine, uric acid, WBC and prolactin were in the normal range; C-reactive protein was detected <5 mg/ml. The plasma concentration of valproic acid was 82.4 mg/l. Urine screens for illicit drugs and blood alcohol concentrations were negative. Urine myoglobine test was not performed.

Quetiapine was discontinued, while medication with valproic acid was not stopped. Within 7 days the clinical symptoms subsided and the pathological laboratory parameters remitted.

We can assume that quetiapine caused this rhabdomyolysis. Regarding the psychovegetative symptoms, it is possible that

we observed the start of an abortive neuroleptic malignant syndrome induced by quetiapine.

A recent case report (Plesnicar et al.; 2007) considers the risk of rhabdomyolysis with quetiapine especially in overdose and indeed some reports of such cases can be found in the literature (Harmon et al.; 1998; Himmerich et al.; 2006, Pollack and Zbuk; 2000; Smith et al.; 2004). In most reported cases patients also received other drugs at the same time, and it can be speculated that polypharmacy might increase the risk for such side effects. In our case the patient was comedicated with valproic acid, which is known to be able to increase quetiapine plasma concentrations. As quetiapine and valproic acid are increasingly used off-label in a reasonable attempt to treat conditions like borderline personality disorder, more people are disposed to these medications and even rare side effects, as rhabdomyolysis definitely is, will occur more often and need to be recognized quickly to be resolved without sequelae for these patients. In our case muscle stiffness was the first sign that the medication was not tolerated. A similar case without elevations of creatine phosphokinase was reported by Fountoulakis et al. (2003). On the other hand massive elevations of creatine phosphokinase without clinical symptoms or only minor symptomatology are reported for second generation antipsychotics, namely olanzapine but also quetiapine (Klein et al.; 2006, Marcus et al.; 1999). The first action that should be taken in all these cases of rhabdomyolysis is to discontinue suspicious medication and to monitor the patient until symptoms are remitted, applying additional measures if appropriate to minimize any harm to the patients by the muscular damage. For example in severe cases enforced diuresis and alkalisation of the urine in order to prevent toxic effects of myoglobin to the kidneys can be useful (for a review see Lindner and Zierz; 2003).

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