
Escitalopram-associated rhabdomyolysis

SHORT CASE REPORT

KAMILLA KVIKSTAD MATHISEN

kamilla.kvikstad.mathisen@nordlandssykehuset.no

Department of Child and Adolescent Psychiatry

Nordland Hospital Trust, Bodø

Kamilla Kvikstad Mathisen, specialist in child and adolescent psychiatry and senior consultant.

The author has completed the ICMJE form and declares no conflicts of interest.

RANDOLF HARDERSEN

Department of Clinical Medicine

Nordland Hospital Trust, Bodø

Randolf Hardersen, PhD, specialist in internal medicine and kidney disease, and senior consultant.

The author has completed the ICMJE form and declares no conflicts of interest.

This case history describes a case of rhabdomyolysis resulting from treatment with escitalopram as monotherapy at the recommended dose. This is a rare and little-known side effect of selective serotonin reuptake inhibitors.

An adolescent in his late teens was referred to the outpatient clinic for child and adolescent psychiatry due to suspicion of an eating disorder. He exercised daily and had lost weight as a result of inadequate energy intake. His condition was interpreted as orthorexia nervosa. During treatment at the outpatient clinic for child and adolescent psychiatry his condition improved, but one year later he showed increasing symptoms of depression, with indication for antidepressant drug treatment. He was taking a calcium supplement with vitamin D due to osteopenia but took no other regular medications during the disease course.

The patient initially benefited from 10 mg escitalopram, but his depressive symptoms increased after six months. The dose was then increased from 10 mg to 15 mg daily. As part of the follow-up at the outpatient clinic for child and adolescent psychiatry, blood tests were taken for screening five days after the dose was increased. These revealed significantly elevated creatine kinase (CK) of > 20 000 U/L (Table 1). He reported no muscle pain but was admitted as an emergency patient to the department of clinical medicine, where he was treated for rhabdomyolysis with intravenous fluid and activity restriction. Serum myoglobin, electrolytes and renal function were normal throughout the disease course.

Table 1

Creatine kinase (CK) levels measured from admission until the levels normalised.

	CK levels (reference range 50–400 U/L)
Day 1, at admission	> 20 000
Day 6, at discharge	4 343
Day 10	3 684
Day 30, 11 days after discontinuation of escitalopram	245

Serum concentration of escitalopram was within the reference range. Pharmacogenetic testing showed normal activity of the cytochrome P450 enzyme, which is the main metaboliser of escitalopram (CYP2C19), and normal expression of the serotonin transporter SLC6A4. The CK level fell during the six-day hospitalisation period (Table 1). At discharge it was concluded that orthorexia and severe undernutrition was the reason for the development of rhabdomyolysis. No change was made to the medication. The patient expressed clearly that he had normalised his energy intake and refrained from exercise in the days before admission. His current body mass index was 18 kg/m².

Four days after discharge, the patient still had elevated creatine kinase despite cessation of physical activity. Creatine kinase is halved every 1.5 days (1) when the triggering cause is removed. Other causes therefore needed to be considered. Escitalopram as a possible contributory cause of the rhabdomyolysis was suspected, and the drug was therefore gradually discontinued with a reduction of 5 mg every two days. Eleven days after the last intake of escitalopram, the CK level was within the reference range. The elimination half-life of escitalopram is approximately 30 hours, and considerably longer for its principal metabolites (2). The patient's depressive symptoms increased somewhat, and according to the patient's own wishes these were treated with talking therapy at the outpatient clinic for child and adolescent psychiatry.

Discussion

In Norway, escitalopram is the most widely used antidepressant of the group of selective serotonin reuptake inhibitors (SSRIs) (3). CK elevation and rhabdomyolysis are not discussed in the Norwegian summary of product characteristics for escitalopram (4), but cases of rhabdomyolysis are reported in international reference works and literature (5, 6).

Rhabdomyolysis is characterised by muscle injury. CK levels are typically elevated, in conjunction with findings of myoglobinuria and symptoms of muscle pain. The degree of severity can vary from asymptomatic to life-threatening, with electrolyte imbalance and acute kidney failure.

Rhabdomyolysis has several potential causes: trauma, muscle compression, intense physical exertion, medication (most often statins), infections, electrolyte imbalance and some types of substance abuse (7). The case in question could not be explained by these most well-known factors.

The patient's perspective

When I was admitted I felt that because of my, at times, strained relationship with food and exercise, conclusions were quickly drawn as to the reason behind the rhabdomyolysis. I felt I was not believed when I said that exercise and food intake had been normal in the period before. In many ways I felt mistrusted and accused, and that was not a good feeling in a vulnerable, serious situation.

Rhabdomyolysis as an adverse effect of low-dose escitalopram in monotherapy was described in 2011 (8). Case studies have described rhabdomyolysis as a result of escitalopram or other SSRIs, but at a higher dosage in those cases (9), in combination with other drugs (10, 11), following overdose (12, 13), or after intense physical activity in combination with a recent increase in dosage (14).

The mechanism behind SSRI-associated rhabdomyolysis is not fully understood. One theory is that serotonin plays a role in the contraction and relaxation of the musculoskeletal system, and that use of SSRIs can potentially lead to prolonged muscle contraction and thereby to rhabdomyolysis in predisposed patients (14). Sensitivity to adverse effects from psychopharmaceuticals is individual, and such effects can have a dose-response relationship.

Rhabdomyolysis resulting from monotherapy with SSRIs is a rare and sparsely documented adverse effect but can potentially be a serious condition. This case study illustrates the importance of close follow-up of possible adverse effects at the start of treatment with SSRIs, when the dose is increased, or when the treatment is long term.

The patient has consented to the publication of this article.

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