Letter to the Editor

Diabetic ketoacidosis after self-poisoning with bromadiolone

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A 54-year-old man, with no medical history, was admitted to the Emergency Department reporting self-poisoning with bromadiolone (estimated quantity 7.5 mg), 4 days prior to admission. During the last 24 h, the patient reported at least twenty episodes of non-bilious vomiting, nausea, weakness and agitation. He was tachypneic (respiratory rate: 32 breaths/min), with a Kussmaul-like breathing pattern. There were no signs of automatic external hemorrhage, due to the ingestion of superwarfarin. The rest of the clinical examination was unremarkable. The electrocardiogram revealed the presence of atrial fibrillation with a rapid ventricular response, while air blood gases were indicative of metabolic acidosis with a high anion gap (pH: 7.18, HCO_3: 5.1 mmol/l, anion gap: 22). Lactic acid levels were within normal limits. The blood glucose value was 492 mg/dl, while presence of ketones in urine examination confirmed the diagnosis of diabetic ketoacidosis. The coagulation profile of the patient was abnormal (INR: 4.76, prolonged pT and apTT). An urgent abdominal computed tomography (CT) scan was ordered in order to exclude the presence of acute hemorrhagic pancreatitis, leading to diabetic ketoacidosis. Computed tomography scan revealed the presence of pancreatic atrophy, probably due to his 20-year history of alcohol abuse, which was terminated 5 years ago. No pathology was depicted on brain and chest CT scan. Besides the application of the treatment protocol for diabetic ketoacidosis, the patient was also administered vitamin K1 and fresh frozen plasma. The metabolic disturbance was resolved, but the patient developed severe hypophosphatemia (P: 0.5 mg/dl) and hypocalcaemia (Ca: 7.6 mg/dl) during treatment, while the rest of electrolyte values were within normal limits, making intravenous correction of hypophosphatemia unfeasible. *Per os* administration of phosphorus with monitoring of calcium, phosphorus, magnesium and potassium values was the treatment of choice. The glycosylated hemoglobin value was 7.1%. The patient was finally discharged 4 weeks later in good general condition. During his follow-up, he remains asymptomatic, with sufficient glycemic control, without initiation of insulin treatment, and free of coagulation disturbances or electrolyte abnormalities. He is being prescribed metformin 1000 mg twice daily, along with his psychiatric treatment (antidepressant therapy with escitalopram, 20 mg once daily). As far as his atrial fibrillation episode is concerned, 6.25 mg carvedilol twice daily was prescribed, along with 2500 IU of tinzaparin per day. This was the initial atrial fibrillation therapeutic approach, since it is not possible to prescribe novel oral anticoagulants to someone who has received bromadiolone for a period of at least 2 months.

Bromadiolone is a superwarfarin, strong and long-acting (with a mean half-life of 20–30 days), leading to greater absorption and coagulation inhibition compared with warfarin [1]. It is lipid soluble with high affinity to liver tissue and slow elimination; thus, self-poisoning with this substance usually requires long hospitalization and close monitoring of the patient [2]. The main clinical manifestations instead of symptoms include bleeding events and gastrointestinal symptoms [3]. In our case, there were no bleeding events, either clinically manifested or documented during physical or paraclinical examination, whereas gastrointestinal symptoms were intense and possibly led to diabetic ketoacidosis on the background of non-diagnosed diabetes mellitus. Diabetic ketoacidosis after self-poisoning is a rare manifestation, previously reported after consumption of pesticides [4]. Hyperglycemia has been reported previously in animal studies after rodenticide intoxication [5]. However, it is unclear to what extent bromadiolone contributed directly to the manifestation of diabetic ketoacidosis. We should have in mind the possible contribution of the early fall of protein C levels due to superwarfarin action. The latter may have led to even further pancreatic ischemia, which in due course may have contributed to the manifestation of bromadiolone-related diabetic ketoacidosis. This is the first report of diabetic ketoacidosis early after bromadiolone self-poisoning on the background of undiagnosed diabetes mellitus.

Conflict of interest

The authors declare no conflict of interest.

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