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Metformin Intoxication, Lactic Acidosis and Death: Case Report

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Abstract: Metformin is an oral antidiabetic agent used as in the treatment of T2DM. A fearsome complication of the use of metformin is lactic acidosis. A nineteen-year-old woman was unconscious and cyanotic in her home. It has been found that about 50 grams of metformin is taken for the purpose of suicide. Glaskow Coma Scale 4/15, Blood Pressure: 70 / 30mmHg, lactic acidosis was detected. Despite the hemodialysis procedure, the patient passed away. Metformin, the keystone of diabetes treatment, inhibits gluconeogenesis, improve insulin resistance, and is excreted in the kidneys without being metabolized. Metformin rarely makes lactic acidosis. High doses of metformin and renal impairment facilitate the formation of lactic acidosis. There is a direct relationship between the degree of lactic acidosis and mortality. Lactic acidosis is thought to be due to the accumulation of lactate by the inhibition of gluconeogenesis. Metformin intoxication may kill the patient through lactic acidosis.

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INTRODUCTION

Metformin is usually used the first drug in treatment type 2 diabetes mellitus. The most frightening complication of the use of metformin is lactic acidosis(Bailey. et al. 1996). We aimed to share the characteristics of our first patient who was died due to severe lactic acidosis due to metformin poisoning.

CASE

A 19-year-old woman was unconscious and cyanotic at home. Observed blood glucose value: 32 mg / dl was detected in the patient who received intravenous glucose. His anamnesis from his family revealed that his consciousness was open 6 hours ago and about 50 missing from the metformin 1000 mg tablet in the medicine box next to him. It has been determined that he did not take any other medication.

Glaskow Coma Scale 4/15, Blood Pressure: 70 / 30 mmHg, Pulse Rate: 55 / min / rhythmic Glucose: 399 mg/dl, Urea: 16mg/dl, Creatinine: 1.92 mg/dl, Na: 147 mmol / L, K: 4.2 mmol / L, pH: 6.8 (7.35-7.45), PO2:93.2 (80-100), HCO3: 4.9 (18-22), Lactate:30 (0.3-0.8). Hemodialysis was performed after the patient was started with colloid and inotropic blood pressure of 90/60 mmHg. Cardiac enzymes were normal. Hemodialysis was not completed due to deep hypotension. The patient continued lactic acidosis in control blood gas and continued inotropic support. The patient who had cardiac arrest at 7th hour of hospitalization did not respond to resuscitation.

DISCUSSION

Metformin, the keystone of diabetes treatment, inhibits gluconeogenesis, breaks insulin resistance and is excreted in the kidneys without being metabolized(Vengerovsky et al. 2016). Metformin rarely makes lactic acidosis. High doses of metformin and renal impairment facilitate the formation of lactic acidosis. There is a direct relationship between the degree of lactic acidosis and mortality (Eppenga et al.2014). Half of patients with lactic acidosis are die. Although the mechanism of lactic acidosis is not fully understood, it is thought to be due to the accumulation of lactate by inhibition of gluconeogenesis (Graham et al. 2011). Hypoglycaemia can also occur in metformin poisoning (Al-abri et al. 2013).

In conclusion we have observed that metformin in a young patient who was previously healthy leads to death by making severe lactic acidosis.

Patients with metformin-induced lactic acidosis should be cautious for hypoglycemia.
Severe lactic acidosis can lead to death.

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