

Acute hyperkalemia induced by hyperglycemia in non-diabetic patient

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Hyperglycemia can induce hyperkalemia in diabetic patients with insulin deficiency [1,2]. It is thus recommended to monitor potassium concentrations in diabetic patients. However, in healthy individuals, acute hyperglycemia appears to lower or not affect the serum potassium concentration [3] and hyperkalemia rarely occurs due to the kidney's capacity to excrete potassium. In this case, we describe a non-diabetic patient who experienced acute severe hyperkalemia induced by transient hyperglycemia during elective surgery.

A 49-year-old woman was admitted to hospital due to lipoma on the abdomen and on the back and the fatty abdomen. The patient had a history of hypertension, treated with angiotensin-converting enzyme inhibitors (ACEi), for 5 years. Her random blood glucose level was 121 mg/dl. The pre-operative fasting glucose level was 109 mg/dl, the serum K⁺ level was 4.5 mmol/L, BUN was 19.3 mg/dl, Cr was 0.7 mg/dl, and urine ketones were negative. After excision of the lipoma over 2 h, the surgeon began abdominal liposuction. Approximately 20 min later, a T wave taller than the accompanying QRS complexes was observed on the ECG. The blood pressure was 110/60 mmHg, and the pulse rate was 61 beats/min. The blood loss was less than 50 ml, and 760 ml of lactated ringer's solution was infused. There was no sign of dehydration. We performed an immediate arterial blood analysis that revealed a K⁺ level of 8.07 mmol/L, Na⁺ 140 mmol/L, pH 7.284, PaCO₂ 33.6 mmHg, PaO₂ 201.4 mmHg, HCO₃ 15.6 mmol/L, and an O₂ saturation of 99.3%. The blood sugar level was 327 mg/dl. We injected calcium gluconate (300 mg) and regular insulin (5 units) intravenously and began an infusion of 50% dextrose solution mixed with regular insulin

(25 units). The tall T waves disappeared within 5 min and the patient was hemodynamically stable. Repeated arterial blood analysis showed K⁺ levels of 5.16 mmol/L, Ca²⁺ 0.95 mmol/L, pH 7.366, PaCO₂ 44.1 mmHg, PaO₂ 201.8 mmHg, HCO₃ 24.7 mmol/L, O₂ saturation 99.0%, and a blood glucose level of 147 mg/dl. The operation proceeded uneventfully, and finished within 3 h. The patient recovered with no specific complication. In the post-anesthetic care unit, she was hemodynamically stable, the serum K⁺ level was 3.9 mmol/L, and urine ketones were negative. She did not complain of any symptom, such as abdominal pain, nausea, vomiting, drowsiness, or weakness. She did not have the previous symptoms of polyuria, polydipsia, fever, chest pain, shortness of breath, or weight loss. The postoperative BUN was 17 mg/dl, and the Cr was 0.6 mg/dl. The serum K⁺ level was 4.4 mmol/L on the day after operation and 4.2 mmol/L on the following day. The blood sugar levels and K⁺ levels were maintained within the normal range. The highest random blood glucose level was 147 mg/dl and the HbA1c was 5.3%. The patient was discharged on the 6th post-operative day, with no complication.

Two major causes of hyperkalemia exist. The first is an altered internal potassium balance, including acidosis, insulin deficiency, hypoaldosteronism, and cell necrosis. The second is an altered external balance, including the effects of ACEi. Hyperkalemia can also occur as a response to blood cell lysis.

It is well-documented that the buffering of excess hydrogen ions in cells leads to potassium movement into the extracellular fluid, to maintain electroneutrality. This is true in metabolic acidosis, caused by the accumulation of mineral acids, but

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is less likely to occur in organic acidoses, such as diabetic ketoacidosis (DKA) [2]. Although, we could not diagnose the patient as having DKA, the acidemia would not explain the severe hyperkalemia noted in this patient.

In cases of severe volume depletion, the ability to handle a potassium load is impaired due to decreased distal fluid delivery, which can diminish potassium secretion [2]. In our case, the patient did not display any sign or symptom of hypovolemia. No evidence of hypoaldosteronism was observed.

The administration of an angiotensin-converting enzyme inhibitor can limit aldosterone release, aggravating hyperkalemia. These drugs can reduce the concentration of circulating angiotensin II and diminish intra-adrenal angiotensin II, which can mediate part or most of the stimulating effect of hyperkalemia [2]. However, this was not true in the present case, as the patient's plasma creatinine and urea levels were normal, and the potassium, chloride and bicarbonate levels were restored to normal [2,4].

We thus conclude that the hyperkalemia that developed during surgery was induced by hyperglycemia in this case. Mild-to-moderate hyperkalemia is common in patients with hyperglycemic crises, such as in DKA. When circulating insulin is low, as in DKA, K^+ is released from cells, raising the plasma potassium levels [2]. Furthermore, an elevation in plasma osmolality causes osmotic water movement from the cells into the extracellular fluid, which is paralleled by K^+ release from the cells. While the cell necrosis induced by the liposuction

process should be considered, we are aware of no report of hyperkalemia during or after liposuction and this therefore seems unlikely to explain the hyperglycemia observed [5].

In conclusion, our case report presents a non-diabetic patient who, during a minor operation, became severely hyperkalemic, apparently induced by hyperglycemia. Because of its potentially fatal consequences, including skeletal muscle weakness and cardiac manifestations, hyperkalemia exceeding 6 mEq/L should always be treated. We emphasize that acute hyperkalemia can occur in various circumstances, as observed in this unpredictable case, and should thus be monitored carefully.

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