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Teaching Point
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Hypertensive crisis precipitated by insulin-induced hypoglycemia with end-stage renal failure

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Hyperkalemia is a medical emergency that can induce deadly cardiac arrhythmias. Treatment of hyperkalemia can be thought of in three distinct steps. First, antagonize the effects of hyperkalemia at the cellular level (membrane stabilization) by administering calcium gluconate; secondly, decrease serum potassium by promoting potassium into cells through co-administration of glucose and insulin and thirdly, remove potassium from the body by dialysis or treatment with potassium-binding resins. The use of insulin to promote the movement of potassium into cells in patients with end-stage renal failure and hyperkalemia is widely supported. Administration of insulin with glucose causes the potassium to fall rapidly; however, hypoglycemia has been noted as a frequent complication [1]. Blood pressure (BP) elevation secondary to hypoglycemia has been demonstrated through the activation of the sympathoadrenal system [2]. Herein we present the case of a patient with end-stage renal failure who developed a hypertensive crisis in the setting of insulin-induced hypoglycemia following treatment for hyperkalemia.

Case report

A frail 71-year-old man with end-stage renal failure on maintenance hemodialysis presented with a thrombosed arteriovenous fistula. His medications included irbesartan HCT 300/12.5 mg daily and moxonidine 0.2 mg mane. Serum potassium (Se K^+) on arrival was 7.0 mmol/L (3.5–5.0 mmol/L); BP was recorded at 170/90 mmHg. Electrocardiogram did not reveal any acute T-wave changes. He was treated overnight with insulin (actrapid 5 units) and 25 mL of 50% glucose. Within 2 h, he was noted to be sweaty and anxious with a blood sugar level (BSL) of 1.3 mmol/L and BP 209/109; Se K^+ 5.0 mmol/L. He was treated symptomatically with a glucose bolus. Whilst awaiting surgical thrombectomy, his Se K^+ rose to >6 mmol/L and he was again administered insulin (actrapid 10 units) and 50 mL of 50% glucose in addition to calcium gluconate. Over the next 2 h, his BP climbed progressively to 270/118 mmHg. The patient complained of a headache and was noted to be confused and drowsy. His BSL was 1.8 mmol/L. A dextrose infusion was commenced with a subsequent fall in BP and rise in BSL (Figure 1). Urgent dialysis was arranged via a temporary vascular catheter.

Discussion

Movement of serum potassium into cells is most frequently achieved by giving insulin. Insulin stimulates the Na-K ATPase pump that moves potassium intracellularly in exchange for sodium in a 2:3 ratio and this effect is independent of insulin's effect on glucose (reviewed in [3]). In the setting of hyperkalemia, 10 units of quick-acting insulin is given often co-administered with 50 mL of 50% dextrose. The effect of insulin is usually seen within 10–20 min and can be expected to reduce the serum potassium by 0.6–1.0 mmol/L. If the patient is already hyperglycemic, supplemental glucose is not required; however, controversy regarding treatment of non-diabetic patients with glucose alone exists [4]. As up to 37% [5] of patients on hemodialysis may have unrecognized abnormalities in glucose tolerance, some patients may not achieve the levels of insulin required in response to a high glucose load [4]. Furthermore, such relative hypoinsulinemia may promote a shift of potassium out of the cell causing a further rise in serum potassium [6].

A close temporal relationship between hypoglycemia and BP has been demonstrated in patients with diabetes [7]. Early reports of hypoglycemia-induced hypertension were observed in diabetic patients treated with propranolol [8] or metoprolol [9] where the clinical picture was one of unopposed α-adrenergic vasoconstriction. More recently, hypoglycemia-induced hypertension has been observed in diabetic patients not on such agents [7]. Severe hypoglycemia selectively stimulates the adrenal medulla and causes a marked release of catecholamines [10]. Angiotensin II facilitates adrenal release during insulin-induced hypoglycemia through binding of both the angiotensin I (AT_1R) and II (AT_2R) receptors. In rats,

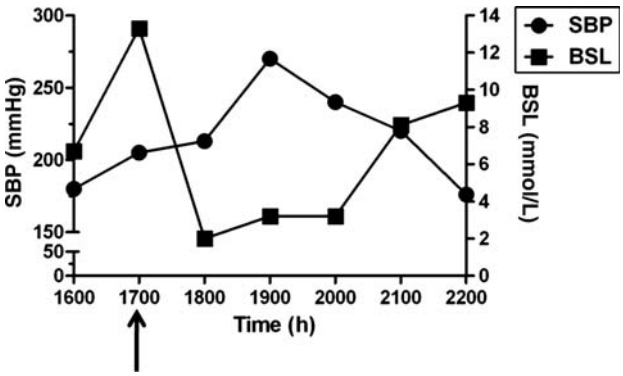

Fig. 1. Systolic blood pressure (SBP) and blood sugar level (BSL) following administration of 50 mL of 50% glucose and 10 units of short-acting insulin (arrow).

blockade of AT_1R alone with losartan did not affect adrenalin release. Although blockade of the AT_2R, the predominant receptor in the adrenal medulla attenuated the response, the greatest effect was achieved with combined blockade of both AT_1 and AT_2R [11].

We observed a striking sympathoadrenal response in our patient despite therapy with irbesartan HCT, a selective AT_1R inhibitor. Classic symptoms of sympathetic nervous system activation were not prominent possibly due to the concurrent use of moxonidine, an imidazole receptor agonist with central sympathetic inhibitory properties. Further moxonidine is known to improve insulin sensitivity in insulin-resistant hypertensive patients [12] and may have enhanced the susceptibility to hypoglycemia in this patient.

Conclusions

Hyperkalemia in patients with end-stage renal failure is a common medical emergency. Evidence supports the use of insulin with glucose as first-line therapy to induce a shift of potassium into the intracellular compartment. Caution is required however as insulin-induced hypoglycemia is common and can cause a rise in BP via stimulation of the sympathoadrenal axis. Blood glucose monitoring with or without supplementary parenteral glucose is therefore essential.

Teaching points

1. Insulin-induced hypoglycemia is common
2. Severe hypoglycemia can precipitate a hypertensive crisis
3. Close monitoring for hypoglycemia is required, particularly following the administration of insulin to non-diabetic patients.

Conflict of interest statement. None declared.

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