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

Lavinia Spain, Peter Iser, Adam Steinberg and Karen M. Dwyer

Department of Nephrology, St. Vincent’s Hospital Melbourne, Fitzroy, Victoria, Australia

Correspondence and offprint requests to: Karen Dwyer; E-mail: Karen.dwyer@svhm.org.au

Keywords: hyperkalemia; hypertension; insulin-induced hypoglycemia

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.

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 (AT1R) and II (AT2R) receptors. In rats,
blockade of AT1R alone with losartan did not affect adrenalin release. Although blockade of the AT2R, the predominant receptor in the adrenal medulla attenuated the response, the greatest effect was achieved with combined blockade of both AT1 and AT2R [11].

We observed a striking sympathoadrenal response in our patient despite therapy with irbesartan HCT, a selective AT1R 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.

References

1. Blumberg A, Weidmann P, Shaw S et al. Effect of various therapeutic approaches on plasma potassium and major regulating factors in terminal renal failure. Am J Med 1988; 85: 507–512
2. Sommerfield AJ, Wilkinson IB, Webb DJ et al. Vessel wall stiffness in type 1 diabetes and the central hemodynamic effects of acute hypoglycemia. Am J Physiol Endocrinol Metab 2007; 293: E1274–E1279
3. Parham WA, Mehdirad AA, Biermann KM et al. Hyperkalemia revisited. Tex Heart Inst J 2006; 33: 40–47
4. Kamel KS, Wei C. Controversial issues in the treatment of hyperkalaemia. Nephrol Dial Transplant 2003; 18: 2215–2218
5. Bergrem HA, Valderhaug TG, Hartmann A et al. Undiagnosed diabetes in kidney transplant candidates: a case-finding strategy. Clin J Am Soc Nephrol 2010; 5: 616–622
6. Conte G, Dal Canton A, Imperatore P et al. Acute increase in plasma osmolality as a cause of hyperkalemia in patients with renal failure. Kidney Int 1990; 38: 301–307
7. Feldman-Billard S, Massin P, Meas T et al. Hypoglycemia-induced blood pressure elevation in patients with diabetes. Arch Intern Med 2010; 170: 829–831
8. Mann SJ, Krakoff LR. Hypertensive crisis caused by hypoglycemia and propranolol. Arch Intern Med 1984; 144: 2427–2428
9. Shepherd AM, Lin MS, Keeton TK. Hypoglycemia-induced hypertension in a diabetic patient on metoprolol. Ann Intern Med 1981; 94: 357–358
10. Cryer PE. Physiology and pathophysiology of the human sympathoadrenal neuroendocrine system. N Engl J Med 1980; 303: 436–444
11. Worck RH, Frandsen E, Ibsen H, Petersen JS. AT1 and AT2 receptor blockade and epinephrine release during insulin-induced hypoglycemia. Hypertension 1998; 31(Pt 2): 384–390
12. Haenni A, Lithell H. Moxonidine improves insulin sensitivity in insulin-resistant hypertensives. J Hypertens Suppl 1999; 17: S29–S35

Received for publication: 29.1.12; Accepted in revised form: 20.4.12
Author/s: Spain, L; Iser, P; Steinberg, A; Dwyer, KM

Title: Hypertensive crisis precipitated by insulin-induced hypoglycemia with end-stage renal failure.

Date: 2012-08

Citation: Spain, L., Iser, P., Steinberg, A. & Dwyer, K. M. (2012). Hypertensive crisis precipitated by insulin-induced hypoglycemia with end-stage renal failure. Clin Kidney J, 5 (4), pp.362-363. https://doi.org/10.1093/ckj/sfs053.

Persistent Link: http://hdl.handle.net/11343/270432

File Description: Published version

License: CC BY-NC