Aldosterone excess: a rare non-nephropathic cause of hypertension in type I diabetes

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Summary
The aetiology of hypertension in type I diabetes is commonly due to the presence of diabetic nephropathy. A rare case of hypertension in a patient with type I diabetes and no proteinuria is reported, where the investigation of borderline hypokalaemia allowed us to make a diagnosis of hyperaldosteronism due to bilateral adrenocortical hyperplasia. Secondary causes of hypertension should always be considered in all diabetic patients, particularly in the absence of clinical proteinuria.

Keywords: aldosterone; nephropathy; hypokalaemia; diabetes

Hypertension in long-term type I diabetes is commonly due to diabetic nephropathy. With increased duration of the diabetic state, there is an increased prevalence of diabetic nephropathy which is often associated with retinopathy. In the absence of retinopathy, secondary causes of the hypertension other than diabetic nephropathy are usually considered to be aetologically important. Aldosterone excess as a secondary cause of hypertension in the non-diabetic population is not uncommon, while in diabetic patients this has not been previously reported.

Case report
A 54-year-old Caucasian man without a family history of diabetes or hypertension, had presented 17 years previously with classical type I diabetes. At diagnosis, his blood pressure was normal at 130/70 mmHg. Four years later, hypertension was diagnosed, with several blood pressure readings greater than 180/100 mmHg. There was no evidence of diabetic retinopathy or nephropathy (no dipstick proteinuria). Renal function and electrolytes were normal; HbA1c was 10.0%. Monotherapy with nifedipine (Adalat LA 20 mg bid) was ineffective while combination with enalapril (Innovace 10 mg od) had only a minimal blood pressure lowering effect. A borderline hypokalaemia (potassium 3.0 mmol/l) was confirmed on biochemical screening. Subsequent investigations included a mineralocorticoid profile after four weeks enalapril withdrawal (box 1). A selenocholesterol scan showed normal to high activity in both adrenal glands, while magnetic resonance imaging revealed bilateral adrenal gland enlargement.

These results confirmed the diagnosis of hyperaldosteronism to be due to bilateral adrenal hyperplasia. Spironolactone was commenced (in addition to the combination antihypertensive therapy) which was effective in normalising the blood pressure (to less than 140/90 mmHg) and potassium (4.3 mmol/l).

Discussion
Hyperaldosteronism associated with type I diabetes has not been previously reported. To our knowledge, this is the first case of bilateral adrenocortical hyperplasia in a patient with type I diabetes.

Aldosterone excess accounts for less than 0.5% of secondary causes of hypertension in the general population; the majority of cases (60%) are due to a single adenoma (Conn’s syndrome), and only 30% are due to bilateral adrenocortical hyperplasia. Aldosterone-producing carcinoma and glucocorticoid-suppressible hyperaldosteronism are extremely rare clinical entities. Sporadic cases of ectopic aldosterone secretion (extra-adrenal Conn’s syndrome) by ovarian tumours have also been reported. Hypokalaemia was the diagnostic clue in the present case, although hyperaldosteronism has been reported in normokalaemic individuals.

Anatomical identification of the cause of aldosterone excess is important to guide appropriate therapy. While medical therapy alone is usually the treatment of choice for bilateral adrenocortical hyperplasia, surgery is required for adrenal adenoma.

In the aetiology of hypertension in type I diabetes, as opposed to type II diabetes, clinical nephropathy is considered to be the major factor. Nephropathy occurs in approximately
Learning points

- In the absence of proteinuria, secondary causes of hypertension should be considered in type 1 diabetes.
- Secondary causes of hypertension should always be suspected in resistant hypertension.
- Full biochemical screening is important in the diagnosis of endocrine hypertension.
- Hyperaldosteronism can present as mild hypokalaemia or even normokalaemia.

Box 2

30% of all diabetic subjects. It is well established that microvascular complications (nephropathy and retinopathy) increase with the duration of diabetes. It was of interest that, in this hypertensive patient who had had type 1 diabetes for more than 15 years, there was no clinical evidence of retinopathy or nephropathy (dipstick evidence of proteinuria). The absence of these clinical features also raised the possibility of a non-nephropathic cause of the raised arterial blood pressure. This case additionally highlights the need to investigate further patients who respond poorly to combination antihypertensive therapy. A full biochemical screen to exclude secondary causes of hypertension should be considered in all patients with type I diabetes, particularly in those without diabetic nephropathy.

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