Diabetes care: can QOF miss the point? Unexpected improvement in diabetes control should prompt a search for an underlying cause

Abstract
The Quality and Outcomes Framework for diabetes mellitus has led to an improvement in diabetes management since its introduction in 2004. However, the focus on reduction of HbA1c must not detract from a holistic approach to patient care.

We present the case of a patient whose unexpected decline in HbA1c levels culminated in an emergency presentation to hospital, where Addison’s disease was diagnosed. Features of adrenal insufficiency were present prior to acute admission.

We review the presenting features of Addison’s disease and discuss the differential diagnosis of reduced HbA1c in diabetic patients. Copyright © 2013 John Wiley & Sons.

Key words
diabetes mellitus; Addison’s disease; adrenal insufficiency; eosinophilia; QOF

Introduction
In 2004, the Quality and Outcomes Framework (QOF) for diabetes mellitus introduced a points system for attainment of target HbA1c values for patients in primary care. These points contribute to practice remuneration for provision of diabetes care, and diabetes management has improved as a result. However, while patients with poor glycaemic control will be identified and offered treatment advice, those with unanticipated reduction in HbA1c may not be brought to the attention of health care professionals as they appear to ‘meet’ QOF targets.

We present a patient with poorly controlled type 2 diabetes in whom Addison’s disease caused recurrent hypoglycaemia and a fall in HbA1c into the non-diabetic range over a year. Suggestive symptoms were present some months prior to her emergency presentation, but a cause for the low HbA1c and hypoglycaemia had not been sought.

This case illustrates the need to beware the patient in whom a falling HbA1c is unexplained, and seek an underlying cause.

Case report
A 71-year-old woman presented to the medical assessment unit with an episode of hypoglycaemia (capillary blood glucose 2.6mmol/L). She gave a two-week history of poor appetite, vomiting and general malaise. On direct questioning, the patient disclosed significant weight loss of 20kg plus recurrent hypoglycaemic episodes over the preceding 12 months, despite a reduction in her gliclazide dose by her GP. She had attended the surgery regarding brown discolouration of the skin on her elbows, for which no explanation was found. She denied recent steroid therapy. Her past history included a nine-year history of type 2 diabetes, hyperlipidaemia, glaucoma and hypothyroidism. She had recently been referred to haematology regarding persistent lymphocytosis but had failed to attend.

Her treatment consisted of metformin 850mg twice daily, gliclazide 80mg once daily, levethyroxine 50µg once daily, and dorzolamide eye drops. She was a non-smoker and drank little alcohol.

On examination she was emaciated and hyperpigmented, particularly in the skin creases (see Figure 1). Her pulse was 60 beats per minute, blood pressure was 108/63mmHg, and temperature was 35.6°C.

Blood tests revealed deranged electrolytes (sodium 133mmol/L [normal range 135–145], potassium 6.2mmol/L [3.5–5.3]) plus a normocytic anaemia (haemoglobin 9g/dl). A diagnosis of acute adrenal failure was made. In view of the past history of autoimmune endocrine...
disease, Addison’s disease (primary adrenal insufficiency) was thought to be the likeliest cause. Blood was taken for cortisol, adrenocorticotropic hormone, and HbA1c levels followed by immediate treatment with intravenous saline, dextrose, and hydrocortisone. The random cortisol level was 12nmol/L (normal range 250–650nmol/L for morning samples); ACTH was >1250ng/L (normal range <46ng/L); and HbA1c 26mmol/mol (DCCT 4.5%) – normal IFCC range for our hospital 20–42mmol/mol (DCCT 4–6%). CT adrenals showed bilateral small glands with no other abnormal morphology.

The patient made excellent progress and was duly switched to oral hydrocortisone 10mg three times daily, plus fludrocortisone 100µg once daily. Her diabetes control deteriorated and she eventually required insulin and metformin to control her diabetes. Her weight improved by 11kg over the subsequent five months.

A retrospective review of the patient’s blood results showed that, 10 months prior to presentation, HbA1c was 67mmol/mol (8.3%) falling to 33mmol/mol (5.2%) two months before admission. Figure 2 illustrates the changes in HbA1c pre- and post-treatment.

Tests one month prior to admission also revealed a lymphocytosis of 4.7x10³ (1–3.8x10³), an eosinophilia of 0.9x10³ (0.0–0.4x10³) and borderline electrolyte derangements, with sodium 132mmol/L and potassium 5.3mmol/L.

**Discussion**

Addison’s disease is rare, but recent studies in primary care suggest the prevalence is higher than previously estimated, at 93–140 per million. Failure of the adrenal glands to secrete sufficient mineralocorticoid and glucocorticoid hormones can present insidiously with non-specific symptoms such as fatigue, weight loss and reduced appetite, or acutely as an adrenal crisis, with shock. Hypoglycaemia can be a feature. In the UK, the most common cause of primary adrenal insufficiency is autoimmune disease, while tuberculosis is the likeliest culprit in developing countries. The key aims of treatment can easily be remembered as ‘the 5 Ss’: Salt, Sugar, Steroids, haemodynamic Support, and a Search for an underlying cause.

As this case illustrates, the presenting features of Addison’s disease can easily be missed. While our patient did have electrolyte derangements prior to presentation, these were mild. Certain characteristic findings, namely hyperpigmentation (particularly of the skin creases, gums and scars), postural hypotension, hypoglycaemia and concurrent autoimmune endocrine disease, may provide diagnostic clues. There may be an eosinophilia and lymphocytosis, as in this case. For this patient, recognition of the pattern of weight loss, hyperpigmentation, hyponatraemia and hyperkalaemia would have allowed earlier commencement of appropriate treatment. Addison’s disease would more usually be linked to type 1 diabetes due to its autoimmune aetiology. Our case had type 2 diabetes but a history of autoimmune thyroid disease.

In the patient with diabetes, an unforeseen reduction in insulin
Diabetes care: can QOF miss the point?

Key points

- Unintentional improvement in diabetes control or unexplained hypoglycaemia are red-flag findings necessitating medical review of the patient
- Beware the patient with type 2 diabetes in whom weight loss is actually unintentional
- Addison’s disease is rare, easily missed, and can present with hypoglycaemia
- Diabetes control can be affected by a myriad of factors from social to psychological to medical, requiring a broad approach on the part of all health professionals

Table 1. Causes which should be considered in the investigation of reduced HbA1c

| Unintentional weight loss |
| Medication issues (e.g. compliance, side effect) |
| Adrenal insufficiency |
| Pituitary insufficiency |
| Coeliac disease, or other causes of malabsorption |
| Renal failure |
| Liver failure |
| Underlying malignancy |
| Insulinoma (very rare) |
| Haemolysis/spurious |

Table 1. Causes which should be considered in the investigation of reduced HbA1c

requirement or HbA1c warrants investigation. Causes which should be considered are listed in Table 1. While weight loss is generally recommended in the treatment of type 2 diabetes, it is crucial to ensure that any sudden change in weight is intentional rather than pathological. While the QOF has undoubtedly had a significant positive effect on diabetes care, a focus on reducing HbA1c levels must not distract clinicians from identifying patients in whom unprecedented normalisation of HbA1c belies serious pathology. We believe that this case serves as a reminder that assessment of the diabetic patient requires a holistic approach, of which biochemical parameters form only a part.

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Declaration of interests

There are no conflicts of interest declared.

References