How can diabetic ketoacidosis save your life?

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A 36-year old gentleman presented with a 2-month history of severe headache, vomiting, polyuria, polydipsia and weight loss. He was dehydrated, tachycardic and hypertensive (220/110mmHg) with hyperglycaemia (32mmol/l) and ketonaemia of 3.3mmol/l and borderline acidosis. He was not previously known to have diabetes. He was treated in line with the local DKA protocol. Investigations for secondary hypertension subsequently showed elevated urinary and plasma normetanephrines. A phaeochromocytoma was biochemically suspected and a CT- abdomen showed a 6cm left retroperitoneal necrotic lesion, for which he underwent laparoscopic left adrenalectomy. Histopathology showed a paraganglioma and genetic testing confirmed an SDHB mutation.

Following recovery from the DKA, a basal-bolus insulin regime was started. When he was alpha and beta blocked, his glycaemic control improved: he stopped his prandial insulin and remained on a low dose of basal insulin. A fasting C-peptide of 89pmol/l and glucose of 7.2mmol/l supported a diagnosis of insulin deficiency, consistent with Type 1 diabetes. However his anti-GAD, anti-IA2 and anti-islet cell antibodies were all negative and his HbA1c was high (11.1%; 98mmol/mol), arguing against a diagnosis of Type 1 diabetes.

Four months after the operation, he developed hypoglycaemia and his insulin was stopped. His HbA1c was in the nondiabetic range (5.7%, 39mmol/mol) and his C-peptide was normal (863pmol/l). A year after his operation his HbA1c increased to 6.8% (50 mmol/mol) and he was found to have a recurrence of his paraganglioma with a new carotid body lesion and possible rib metastasis, suggesting that his diabetes was a result of excess catecholamines.