Hyperglycaemic emergency and type of diabetes mellitus – a case report

RN Oputa, C Ekwueme, P Obi, AC Anyanwu, O Madueke

Abstract
A 23-year-old obese male was admitted with a blood glucose greater than 33.3mmol/l. He was conscious, without ketones in his urine and required large doses of insulin. His illness was precipitated by hot water burns which caused cellulites. Initial C-peptide value was low. Metformin and insulin were started but insulin was stopped after the fourth week after admission, before discharge from hospital. On follow up he had lost weight, his blood glucose normalised on oral medication, and his C-peptide had risen to above the normal range. The case demonstrates difficulties in classification of type of diabetes after acute presentation.

Introduction
Diabetes mellitus is a metabolic syndrome characterised by hyperglycaemia and diverse complications. There is often a long period of absence of symptoms in type 2 diabetes (T2DM), which accounts for up to 90% of all the forms of diabetes.[1-4] Type 1 diabetes (T1DM) characterised by autoimmune damage of pancreatic beta cells, and patients require insulin for treatment. Various markers and factors are implicated in the aetiopathogenesis of diabetes – autoantibodies, Human Lymphocyte Antigen (HLA) alleles, infection, insulin resistance, insulin secretory abnormalities, and alcohol – depending on the type of diabetes.[5-8]

At initial presentation it may be difficult to determine the type of diabetes (T1DM and T2DM).[1-2] We present a 23-year-old male who presented with severe hyperglycaemia, and obesity. His daily insulin requirement was above 120 units, his serum C-peptide level improved and his blood glucose was subsequently well controlled on diet and metformin only.

Case report
A 23-year-old male presented to the Emergency Unit of our hospital with two weeks history of frequent urination and body weakness. One week before he had a domestic

Correspondence to Dr RN Oputa, Endocrine Unit, Department of Internal Medicine, Federal Medical Centre, Owerri, Imo State, Nigeria. Email: regoputa@ yahoo.com . Postal Address: P.O.Box 7249, Aladinma, Owerri, Imo State, Nigeria.

accident with a hot water burn to his lower abdomen and upper thigh, later leading to cellulitis. He was not known to have diabetes but his maternal grandfather and uncle had diabetes.

He was obese, with a body mass index (BMI) of 34.3 Kg/m2, febrile (38.4 o C) and moderately dehydrated. There was a scald on the lower abdomen and upper thigh with signs of cellulitis. A glucose meter reading recorded "HIGH" equivalent to a blood glucose (BG)>33.3mmol/l. Urine was positive for glucose but not ketones.

Investigations revealed a slightly raised white cell count (WCC) of 11,300. Erythrocyte sedimentation rate (ESR) was 37mm/hour. Renal function and electrolytes were normal, as was urine culture. A serum C-peptide level later returned low at 0.5 mg/ml, and a 24 hour urinary cortisol was also normal.

He was treated initially with antibiotics, intravenous normal (0.9%) saline, and hourly low dose insulin. Metformin 1gm bd was also started and he was later transferred to subcutaneous three-times daily soluble insulin and long-acting insulin (glargine) at night. Blood glucose levels were initially high and insulin doses had to be increased to 120 units/day. With intensified dietary advice, and calorie restriction, glycaemic control improved and insulin was withdrawn completely in the fourth week after admission, and three days before discharge.

At out-patient follow-up, two months after discharge, he was on metformin 500mg OD only as well as a 1500 calorie diet. Weight had dropped from 116 kg to 104 kg. A repeat serum C-peptide level at this time was adequate at 2.0mg/ml.

Discussion
There is often a clinical challenge in identifying and classifying a new case of diabetes that presents as an emergency as in this case report. This young obese patient presented with a severe hyperglycaemia and needed high doses of insulin – up to 120 units daily. The C-peptide level on admission was low and could fit with a pattern of T1DM. However, follow up showed that the patient had T2DM, confirmed by a later normal c-peptide level. Serial C-peptide estimation is a useful guide in monitoring pancreatic beta cell function and insulin secretary reserve.[6,9] For T1DM, pancreatic beta cell function is normally destroyed within six to eighteen months of onset of the illness.[10,11]

It has been observed that some factors can temporarily suppress or down-grade pancreatic beta cell function,

such as lipotoxicity and glucotoxicity.[7,8] Other factors are infection, alcohol and herbal medicines.[12-16] The degree of damage to beta cell function will depend on the duration of insult, intensity of pathology, genetic factors, state of nutrition and pre-injury status.[17] This appears to be the case in this patient – as beta cell function soared above normal with the control of infection, weight loss, diet, exercise and oral medication. The current C-peptide level suggests the presence of insulin resistance. Insulin therapy in Type 2 DM during emergency care will help rest the pancreas and aid recovery of beta cell function.[7]

In our patient, c-peptide levels suggested that he was insulin-deficient, with severely reduced beta-cell function, at the time of presentation. However at two-month follow-up his clinical characteristics were of T2DM, and c-peptide level was very adequate and possibly suggestive of insulin resistance. Our case also has similarities, and differences, with the syndrome of "atypical ketosis – prone type 2 diabetes" which has been described in African and African-origin subjects.[18] Like our patient, this syndrome is associated with abrupt-onset in young and sometimes obese subjects. They are hyperglycaemic but also usually ketotic (unlike our patient). Later they improve and usually come off insulin, and may sometimes even revert to normoglygaemic.

In conclusion, the case presented here demonstrates the frequent difficulty in differentiating between T1DM and T2DM when young, and particularly obese, patients present with acute hyperglycaemia. If available, serum c-peptide levels can be helpful; but from a clinical point of view it is always safest to treat initially with insulin, and consider withdrawal of this drug later.

Author declaration

References

1. Boyle JP, Engelgau MM, Thompson TJ, et al. Estimating prevalence of Type 1 and Type 2 diabetes in a population of African Americans with diabetes mellitus. Amer J Epidemiol 1999; 149: 55-65
2. Holman N, Young B, Gadsby R, Current prevalence of Type 1 and Type 2 diabetes in adults and children in the UK. Diabet Med 2015; 32: 1119-1120.
3. De Fronzo RA, Ferrannini E, Simmet P, Alberti KGMM (Eds). International Textbook of Diabetes Mellitus. 4th ed Publ Wiley, Chichester, UK; 2015.
4. Adadevoh BK. ''Temporary Diabetes'' in adult Nigerians. Trans R Soc Trop Med Hyg. 1968; 62: 528-530
5. Winter WE, Maclaren NK, Riley WJ, et al. Maturity – onset diabetes of youth in black Americans. New Engl J Med 1987; 316: 285-291
6. McFarlane SI, Chinken RL, Hirsch S, et al. Near normoglycaemic remission in African – Americans with type 2 diabetes mellitus is associated with recovery of beta cell function. Diabet Med 2001; 18: 10-16
7. Weng J, Li Y, Xu W et al. Effect of intensive insulin therapy on beta-cell function and glycaemic control in patients with newly diagnosed type 2 diabetes: a multicentre randomised parallel-group trial. Lancet 2008; 371: 1753-1760
8. Benerji MA, Chaiken RL, Lebovitz HE. Long-term normoglycaemic remission in black newly diagnosed NIDDM subjects. Diabetes 1996; 45: 337-341
9. Oputa RN, Ekwueme C, Amah JN, Chinenye S, Nwokolo CJ. Connectin Peptide (C-peptide) and the duration of diabetes mellitus amongst patients at the Federal Medical Centre (FMC), Owerri, South East, Nigeria. Res J of Health Sci 2016; 4: 9-14
10. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 2002; 25: S5 – 20.
11. Evans JM, Newton RW, Ruta DA, MacDonald TM, Morris AD. Socio-economic status, obesity and prevalence of type 1 and type 2 diabetes mellitus. Diabet Med. 2000; 17: 478-480
12. Osuntokun BO, Akinkugbe FM, Francis T, et al. Diabetes mellitus in Nigerians: a study of 832 patients. West Afr Med J 1971; 20: 295-312
13. McLarty DG, Diabetes in Africa In: Krall AP, Alberti KGMM, Serrano-Rio M eds. World Book of Diabetes in Practice. Publ: Elsevier, Amsterdam; 1988: pp218-228
14. Oli JM, Nwokolo C. Diabetes after infectious hepatitis: a follow-up study. Brit Med J 1979; 1: 926-931
15. Cole TO, Adadevoh BK. Herbs and diabetes mellitus. Niger Med J 1974; 4: 51-55
16. Dodu SR. Diabetes in the Tropics. Brit Med J 1967; 2: 747-752
17. Nanita R, Abe S, Kibara Y, et al. Insulin resistance and insulin secretion in chronic hepatitis C virus infection. J Hepatol 2004; 41: 132-138
18. Mauvais-Jarvis F, Sobngwi E, Porcher R et al. Ketosis-prone type 2 diabetes in patients of sub-Saharan African origin. Diabetes 2004; 53: 645 653