Changing diabetes®

Errors in diagnosis, classification and coding on diabetes registers

Samuel Seidu MRCGP, MSc (Diabetes), Honorary GP Lecturer and Research Associate, Leicester Diabetes Centre

Diabetes prevalence in the UK is estimated to be approximately 2.9 million (Diabetes UK, 2012). The scale of the diabetes epidemic and its financial implications are already large enough for the NHS. Therefore, if a significant proportion of people in the UK diagnosed with diabetes have been given the wrong diagnosis or classification, it will only serve to make a bad situation worse.

The psychological impact of wrongly labelling someone as having diabetes can be very devastating. Treatment modalities for various types of diabetes are different and should be used appropriately to derive health outcomes, but this can only be achieved if the correct codes are given to the patient at diagnosis or at any point during treatment. A recent pilot study in England (de Lusignan et al, 2010) provided evidence of misclassification, miscoding and, even worse, misdiagnosis. Following that, a "toolkit" for each of the six main GP IT systems was developed to identify those who may be misdiagnosed, misclassified or miscoded, thus improving the diagnosis and coding of diabetes.

Even though type 1 and type 2 diabetes are the most common types of diabetes, with the latter estimated to account for approximately 90% of diabetes cases globally (World Health Organization, 2011), the American Diabetes Association describes many types and subtypes of diabetes, including eight groups under the heading "other types" (American Diabetes Association, 2007).

This complex mode of classification provides a perfect fertile ground for errors in coding and classification. In 2006, when the Quality and Outcomes Framework (QOF) changes were made and doctors were required to report diabetes as type 1 or type 2, a 22% reduction in the number of people on diabetes registers was noted (Hippisley-Cox and O'Hanlon, 2006).

Type 2 diabetes has a genetic propensity that becomes overt as a result of lifestyle changes, such as decreased physical activity and poor diet. It is characterised by insulin resistance with inadequate pancreatic beta-cell insulin secretion to compensate for the insulin insensitivity. Insulin insensitivity is, in turn, generally characterised by obesity and increased intra-abdominal fat.

Even though diabetic ketoacidosis is uncommon in type 2 diabetes, it can present in rare situations, especially in advanced diabetes. Ketosis-prone type 2 diabetes is an atypical diabetes (type 1B diabetes), also called flatbush diabetes, typical in African-Caribbeans (Umpierrez et al, 2006).

Type 1 diabetes usually begins before the age of 40 years, although there are exceptions. It accounts for only about 5–10% of all cases of diabetes (Daneman, 2006). Although environmental factors such as exposure to various viruses have been noted to trigger the onset and progression to overt diabetes, there is a strong genetic component. It is associated with deficiency of insulin due to an autoimmune disorder in which antibodies are produced against the islet cells of the pancreas.

Maturity onset diabetes of the young (MODY) accounts for 1–5% of all cases of diabetes and is usually inherited as an autosomal dominant disease (Fajans et al, 2001). The genetic defect is a mutation in the genes necessary for insulin secretion. People with MODY are usually not diagnosed until later in life, or sometimes they are wrongly diagnosed as having type 1 or type 2 diabetes. They are generally not obese and diagnosis is made usually under the age of 25 years (Fajans et al, 2001).

The solution to the problem of diabetes classification for many patients can now be aided by the use of a pragmatic and simple clinical guideline, summarised in *Figure 1* (adapted from Royal College of General Practitioners, 2011). This can at least serve as a starting point for primary care teams who will then seek expert advice in more challenging cases. ■

Conflict of interest

Dr Samuel Seidu has received honoraria for serving on various Advisory Boards for Novartis, Novo Nordisk and Janssen.

American Diabetes Association (2007) Diagnosis and classification of diabetes mellitus. *Diabetes Care* **30**: S42–7

Daneman D (2006) Type 1 diabetes. *Lancet* **367**: 847–58

De Lusignan S, Khunti K, Belsey J et al (2010) A method of identifying and correcting miscoding, misclassification and misdiagnosis in diabetes: a pilot and validation study of routinely collected data. *Diabet Med* **27**: 203–9

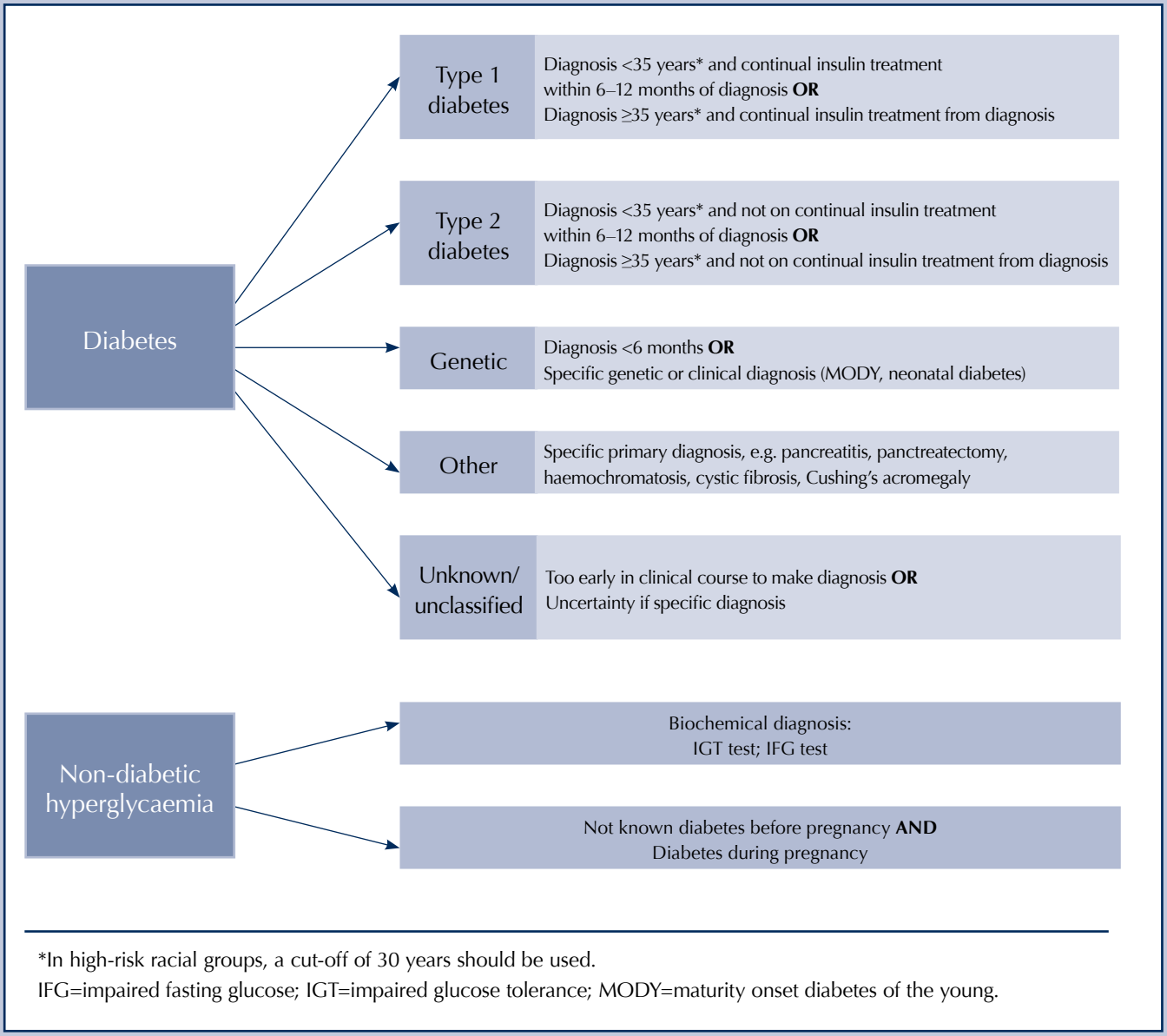

Figure 1. Simple clinical guidelines for diabetes classification (adapted from Royal College of General Practitioners, 2011).

Diabetes UK (2012) *Diabetes in the UK 2012: Key statistics on diabetes*. Diabetes UK, London. Available at: http://bit.ly/MoJ1Nv (accessed 11.02.13)

Fajans SS, Bell GI, Polonsky KS (2001) Molecular mechanisms and clinical pathophysiology of maturity-onset diabetes of the young. *N Engl J Med* **345**: 971–80

Hippisley-Cox J, O'Hanlon S (2006) *Rapid response: Identifying patients with diabetes in the QOF – two steps forward one step back*. BMJ, London. Available at: http://bit.ly/UuKDtX (accessed 11.02.13)

Royal College of General Practitioners (2011) *Coding, classification and diagnosis of diabetes: A review of the coding, classification and diagnosis of diabetes in primary care in England with recommendations for improvement*. NHS Diabetes, Newcastle Upon Tyne. Available at: http://www.diabetes.nhs.uk/document.php?o=208 (accessed 04.03.13)

Umpierrez GE, Smiley D, Kitabchi AE (2006) Narrative review: ketosis-prone type 2 diabetes mellitus. *Ann Intern Med* **144**: 350–7

World Health Organization (2011) *Diabetes fact sheet no. 312*. WHO, Geneva, Switzerland. Available at: http://bit.ly/4VF40h (accessed 11.02.13)