Abstract

Background:

Type 2 diabetes is a heterogeneous disease with multiple causes revolving around beta cell dysfunction, insulin resistance and enhanced hepatic glucose output. Clinical judgement based on obesity status, age of onset and the clinical perception of residual beta cell insulin secretory function (hence insulin-requiring or not), has been used to determine therapeutic choices for each patient. Further laboratory testing of the clinically defined type 2 diabetes unmasks the various aetiologic types within the single clinical group.

Objective:

To determine the aetiological types of the clinically defined type 2 diabetic patients, their chosen therapies at recruitment and the quality of glycaemic control achieved.

Design:

Descriptive cross-sectional study.

Setting:

Diabetes out-patient clinic of Kenyatta National Hospital, Nairobi, Kenya.

Results:

A total of 124 patients with clinical type 2 diabetes were included, 49.2% were males. The mean duration of diabetes in males was 26.09 (20.95) months and that of females was 28.68 (20.54) months. The aetiological grouping revealed the following proportions: Type 1A-3.2%, Type 1B-12.1%, LADA-5.7%, and "true" type 2 diabetes 79.0%. All the patients with Type 1A were apparently, and rightly so, on "insulin-only" treatment even though they did not achieve optimal glycaemic control with HbA1c % = 9.06. However the study patients who were type 1B and LADA were distributed all over the treatment groups where most of them did not achieve optimal glycaemic control, range of HbA1c of 8.46 -10.6%. The patients with "true" type 2 were also distributed all over the treatment groups where only subjects on 'diet only' treatment had good HbA1c of 6.72% but those in other treatment groups did not achieve optimal glycaemic control of HbA1c, 8.07 - 9.32%.

Conclusion:

Type 2 diabetes is a heterogeneous disease where clinical judgement alone does not adequately tell the various aetiological types apart without additional laboratory testing of C-peptide levels and GAD antibody status. This may partly explain the inappropriate treatment choices for the various aetiological types with consequent sub-optimal glycaemic control of those patients.