There really is no such thing as mild diabetes: a new perspective on an old idea

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Although diabetes-related microvascular complications are the commonest cause of blindness, non-traumatic lower limb amputation and renal failure in the Western world,1 macrovascular disease outcomes dominate discussions on type 2 diabetes. Evidence that glycaemic interventions reduce mortality from cardiovascular disease is disappointing.2-4 Studies where beneficial effects on cardiovascular outcomes have been reported focus on non-glycaemic effects in very specific patient groups.5-7

It is argued that the benefits of glycaemic control are small, and guidelines stress an ‘individualised’ approach to HbA1c which, in reality, sanctions acceptance of increased HbA1c values.

This has overwhelmed a previous philosophy ‘that there is no such thing as mild diabetes’, and ignores the relationship between the incidence and impact of microvascular diabetic complications and glycaemic control.2,8

Gray et al,9 using the UKPDS dataset, showed the potential benefits in microvascular complications of improved glycaemic control in newly diagnosed patients with type 2 diabetes. Eastman et al10 described the potential microvascular benefits of achieving near ‘normoglycaemia’ control (HbA1c 55.2 mmol/mol (7.2%)) in a population of patients with type 2 diabetes. This modelling study predicted that, if the whole population achieved an HbA1c of 7.2% (55.2 mmol/mol) there would be a 72% reduction in diabetes-related blindness, 87% reduction in end stage renal failure and a 67% reduction in amputations.

The Core Diabetes Model11,12 has examined the effect of HbA1c targets on the incidence and cost of complications in the USA13,14 and the UK. Baxter et al15 reported the impact of a modest sustained improvement in HbA1c of ~9 mmol/mol (0.8%) in all patients with type 2 diabetes in the UK over 25 years. This modest improvement would prevent 750,000 diabetic microvascular complications and estimated cost avoidance in excess of £4.5 billion.

This study has modelled a population-based incremental improvement in HbA1c and has not adopted a prespecified target HbA1c or targeted ‘high-risk patients. Since 2002 a HbA1c target strategy has been in place. This focuses on people with HbA1c above target who are thought of as ‘high-risk’.16,17 These targets have been adjusted a number of times based on arguments about scientific validity and real-world achievability.18-21 Although targets are an accepted strategic device for improving performance,22 it is also recognised that they can unwittingly create a situation of binary outcome – success and failure – and be perversely counterproductive.23,25

A study using the CPRD primary care database20 showed that 70% of patients with type 2 diabetes where on one oral agent, 33% on two and only 7% on triple therapy. The mean HbA1c in these groups was 68.3 mmol/mol (8.4%), 72.7 mmol/mol (8.8%) and 75 mmol/mol (9%), respectively. This generated the idea of ‘clinical inertia’, which describes the reluctance of clinicians to escalate therapy in the face of suboptimal diabetes control.

However, CSD data on the distribution of HbA1c in the type 2 diabetes population give an alternative perspective to the issue of clinical inertia.27 The data show that the majority of people with type 2 diabetes (59%) have an HbA1c of 59 mmol/mol (7.5%) or below, 30% an HbA1c above 64 mmol/mol (8%) and only 16% above 75 mmol/mol (9%).

These data highlight two important issues:

- An alternative explanation of why inertia is apparently common but may be unrecognised in primary care.
- That there is also a phenomenon of ‘understandable/acceptable’ clinical inertia based on HbA1c targets, and this is potentially much more damaging than what we had previously thought.

Clinical inertia suggests a willful inactivity on behalf of the healthcare professional who fails to escalate therapy when required. However, these data show that a large proportion of the type 2 diabetes population (59%) who would be seen in primary care actually have an HbA1c which would be considered at target (<59 mmol/mol (7.5%)). These patients would currently not be viewed as requiring additional therapy. Current clinical inertia in the real world may therefore be a reflection of the fact that a majority of patients with type 2 diabetes, when judged against accepted HbA1c targets, do not appear to need therapy intensification.

However, Baxter et al also reported that, although the greatest reduction in costs per patient achieved by improving glycaemic control was seen, as predicted, in those people with the highest HbA1c (individual benefit),9,10,28 this high-risk group accounts for less than 30% of the total population and only accounts for 50% of the cost avoidance seen in the whole population.

The other 50% of cost avoidance is actually achieved by reducing (or preventing the rise in) HbA1c in people with HbA1c values below 59 mmol/mol (7.5%). This group actually accounts for the majority (59%) of the type 2 diabetes population and would cur-

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rently be considered at target ‘low risk and safe’. This low-risk at or near target population has become ‘hidden in plain sight’ and the significant contribution to the overall population risk and cost completely overlooked.

The clinical inertia attributed to inaction in people we currently recognise as high-risk may be magnified by the failure to recognise that the majority of patients with type 2 diabetes have HbA1c at or below current targets and, although at low individual risk, make a significant contribution to the total population risk (and costs).

We should consider a strategy based on incremental improvement in HbA1c in all patients with type 2 diabetes by (1) recognising the reality of continuous risk; (2) moving away from dividing the population into those who have failed (and are at risk) and those who succeed and are thought of as ‘safe’; and (3) focusing on the ‘prevention of progression’ and ensuring microvascular risk is recognised and appropriately managed through glycaemic control, as it is clear that there really is no such thing as mild diabetes.

Conflict of interest

RH and MB are employed by Sanofi. The Impact Diabetes programme has been developed in partnership with Diabetes UK, JDRF and Sanofi Diabetes and is funded by Sanofi.

References

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