Clinical Inertia in Type 2 Diabetes Management: Evidence From a Large, Real-World Data Set

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Despite clinical practice guidelines that recommend frequent monitoring of HbA_{1c} (every 3 months) and aggressive escalation of antihyperglycemic therapies until glycemic targets are reached (1,2), the intensification of therapy in patients with uncontrolled type 2 diabetes (T2D) is often inappropriately delayed. The failure of clinicians to intensify therapy when clinically indicated has been termed "clinical inertia." A recently published systematic review found that the median time to treatment intensification after an HbA_{1c} measurement above target was longer than 1 year (range 0.3 to >7.2 years) (3). We have previously reported a rather high rate of clinical inertia in patients uncontrolled on metformin monotherapy (4). Treatment was not intensified early (within 6 months of metformin monotherapy failure) in 38%, 31%, and 28% of patients when poor glycemic control was defined as an HbA_{1c} ≥7% (≥53 mmol/mol), ≥7.5% (≥58 mmol/mol), and ≥8% (≥64 mmol/mol), respectively.

Using the electronic health record system at Cleveland Clinic (2005–2016), we identified a cohort of 7,389 patients with T2D who had an HbA_{1c} value ≥7% (≥53 mmol/mol) ("index HbA_{1c}") despite having been on a stable regimen of two oral antihyperglycemic drugs (OADs) for at least 6 months prior to the index HbA_{1c}. This HbA_{1c} threshold would generally be expected to trigger treatment intensification based on current guidelines. Patient records were reviewed for the 6-month period following the index HbA_{1c}, and changes in diabetes therapy were evaluated for evidence of "intensification" (e.g., increase in OAD dose, addition of another OAD, addition of a glucagon-like peptide 1 receptor agonist, or addition of insulin). As shown in Fig. 1, almost two-thirds of patients had no evidence of intensification in their antihyperglycemic therapy during the 6 months following the index HbA_{1c} ≥7% (≥53 mmol/mol), suggestive of poor glycemic control. Most alarming was the finding that even among patients in the highest index HbA_{1c} category (≥9%

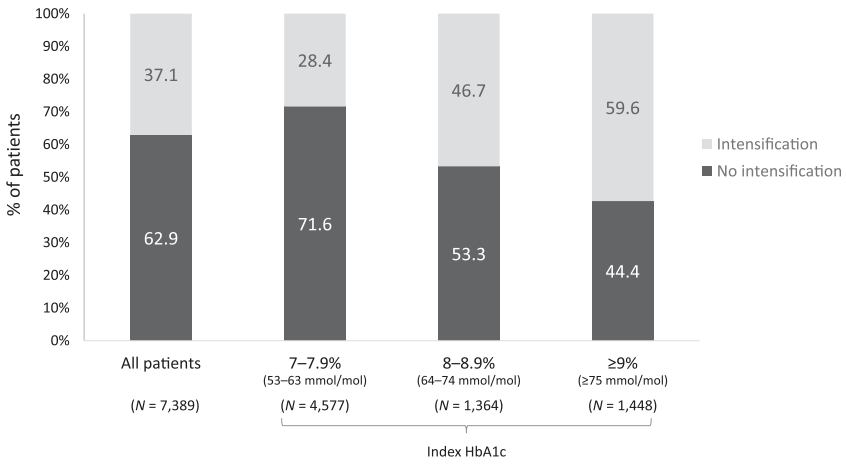

Figure 1—Rates of intensification and nonintensification of antihyperglycemic therapy observed among 7,389 patients with T2D during a 6-month period following an HbA_{1c} ≥7% (≥53 mmol/mol). All patients had been using a stable regimen of two OADs for at least 6 months preceding the index HbA_{1c}.

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[≥75 mmol/mol]), therapy was not intensified in 44% of patients, and slightly more than half (53%) of those with an HbA_{1c} between 8 and 8.9% (64 and 74 mmol/mol) did not have their therapy intensified. Other than perhaps a patient reporting noncompliance with their existing regimen, it is hard to imagine why an intensification of therapy would not occur under these circumstances. Of note, evidence for treatment intensification in this analysis was based solely on physician orders pertaining to antihyperglycemic drugs and did not capture nonpharmacologic forms of therapy intensification (e.g., nutrition or weight management consultation orders); nor did it capture medications prescribed for secondary purposes, such as weight loss, which could be considered a form of intensification.

Unfortunately, these real-world findings confirm a high prevalence of clinical inertia with regard to T2D management. The unavoidable conclusion from these data, which here represent only one institution, is that physicians are not responding quickly enough to evidence of poor glycemic control in a high percentage of patients, even in those with HbA_{1c} levels far exceeding typical treatment targets. Clearly, each patient is unique and there are clinical caveats that limit the generalization of data as presented here. Regardless, the problem of clinical inertia cannot be denied, and every effort should be put forth to manage patients as aggressively as possible to achieve each individual's appropriate glycemic target, which may not always be <7%. We are planning additional analyses to further investigate patterns of specific intensification therapies in this cohort and subsequent changes in HbA_{1c} values, as we suspect less-than-aggressive intensification ("intensification inertia") may also be contributing to suboptimal diabetes management.

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