Optimizing therapeutic options for Hispanic patients with diabetes mellitus and prediabetes: A review of the literature and clinical pearls

On the basis of levels of both fasting blood sugar and glycosylated hemoglobin (HbA1c), the U.S. Centers for Disease Control and Prevention (CDC) estimated that 18.8 million people had diabetes mellitus and 79 million people had prediabetes in the United States in 2010.\(^1\)\(^2\) Diabetes mellitus is the seventh leading cause of death in the United States, as well as the leading cause of nontraumatic amputations, cardiovascular disease, and stroke.\(^1\)\(^2\)
An ominous epidemiologic finding is that the estimated prevalence of diabetes mellitus in individuals aged 18 to 44 years is 11.3%, whereas the estimated prevalence of this disease in the overall population is 8.2%. These statistics suggest a substantial increase in morbidity rates associated with diabetes mellitus in the near future.

As the prevalence of diabetes mellitus grows annually, clinically significant disparities between ethnic groups continue to emerge—especially in regard to Hispanic patients. In 2011, Hispanics’ risk for diabetes mellitus was greater than that of non-Hispanic whites, whether comparing men (Hispanics, 45.4%; non-Hispanics, 26.7%), women (Hispanics, 52.5%; non-Hispanics, 31.2%) or the elderly (Hispanics, 25.8%; non-Hispanics, 15%).

In examining subpopulations, the rate of diabetes mellitus (and, one can hypothesize, prediabetes) is nearly 2.5 times greater for young Hispanics (aged 18-44 years) than young non-Hispanic whites (3.2% vs 1.3%, respectively). The diabetes rate is more than doubled for obese (body mass index [BMI] >30) Hispanics compared to non-obese Hispanics (BMI <25)—15.3% vs 7%, respectively.

In addition, end-organ damage—like renal disease—secondary to diabetes is more prevalent in matched controls of Hispanics vs non-Hispanic whites, at a rate of 3 to 4 times.

Most concerning is the fact that type 2 diabetes mellitus (T2DM) is associated with family history. This familial cycle of disease can be broken through aggressive primary care intervention by optimizing treatment with medications while simultaneously addressing the patient’s weight, physical activity, community support, and education. Family and primary care physicians are uniquely positioned to assume leadership roles in diabetes management teams and to make great strides toward breaking the disease cycle.

To aid in this endeavor, the present article briefly reviews the pathophysiologic characteristics of diabetes mellitus with an emphasis on the mechanisms of action of currently approved medications. The article also reviews programs that are available to assist physicians in caring for Hispanics who have, or who are at risk for, diabetes mellitus.

Pathophysiology review

Type 1 diabetes mellitus

Type 1 diabetes mellitus (T1DM) is a disease of the pancreas caused by β-cell destruction that leads to a complete lack of insulin production and secretion. This loss of insulin, in turn, leads to severe hyperglycemia and ketoacidosis, requiring exogenous insulin treatment (See chart pages 8-9).

Currently, T1DM is more prevalent in pediatric and adolescent populations than is T2DM. Most cases of T1DM can be classified as autoimmune in origin, but a small number of cases are idiopathic. A number of genetic markers have been shown to predict increased risk for T1DM in first-degree relatives of T1DM patients. Specifically, the frequency of β-cell autoimmunity mimics the prevalence of T1DM in populations. There appears to be a decreased frequency of β-cell autoimmunity in Hispanic populations (2.7%) compared to African-American populations (3.3%) and non-Hispanic white populations (3.9%).

T1DM is a chronic disease with a prodromal period of β-cell destruction in the pancreatic islets. There are two subtypes, 1A and 1B. Historically, T1A would have circulating antibodies associated with islet cell destruction whereas T1B (or idiopathic) would not. It was thought that one way in which autoimmune and idiopathic T1DM can be distinguished is through the detection of antibodies to glutamic acid decarboxylase 65 (GAD65) or insulin (insulin autoantibodies). However, inability to detect these GAD65 for instance may simply imply complete destruction of the islet cells and not indicative of etiology.

When autoantibodies to GAD65 or insulin are formed, T lymphocytes are stimulated to begin attacking pancreatic β cells. The CD3 protein, which is part of a receptor found on the T cells, plays a role in the cascade that activates these cells. This protein transduces signals from an antigen receptor as the antigen binds to the T cell. T cells release chemicals, such as interferon-γ and tumor necrosis factor-α, that aid in the destruction of β cells.

Researchers are investigating immunebased approaches to prevent these antibodies from causing the cellular destruction.
that leads to the development of T1DM. At present, however, pharmacologic treatment of patients with T1DM is limited to life-long insulin replacement.

**Type 2 diabetes mellitus**
Type 2 diabetes mellitus is characterized by insulin resistance in muscle, liver and adipose tissue. Mounting evidence suggests a genetic predisposition to the accumulation of liver fat in Hispanics.6-11 These genetic data confirm clinical findings of increased visceral fat, decreased β-cell function, and acute insulin response in overweight and obese Hispanic children.10-12 The liver, as well as muscle and adipose tissues, represent targets for most currently available oral hypoglycemic agents (See chart pages 8-9). Insulin resistance in these tissues initially increases insulin production but eventually leads to β-cell failure and hyperglycemia. Obesity, inactivity, stress and genetic predisposition can all play roles in the development of T2DM.1,2,5

Diagnosis of T2DM is generally based on tests for fasting plasma glucose or oral glucose tolerance. The latter test is less preferred because of uncertainty regarding its reproducibility.13 Testing for HbA1c is the newest diagnostic measure for T2DM. An HbA1c level of 6.5% or more is considered diagnostic of diabetes mellitus, per the 2011 Standards of Medical Care of the American Diabetes Association (ADA).13 Treatment of patients with T2DM often requires multiple drugs, especially in the presence of comorbidities such as hypertension and hyperlipidemia.13

**Insulin resistance and glucose homeostasis**
As glucose is absorbed into the small intestine, multiple pathways are engaged. These pathways are described in the following text, though they do not necessarily occur in the sequence described, nor do they take place in isolation from one another.3,6

Insulin is released from the pancreas, allowing the cells of the body to absorb glucose for energy.3-6 In the liver, insulin stimulates the conversion of glucose into glycogen, for storage, by activating certain enzymes. This stored glycogen is broken down to glucose in times of fasting or low blood sugar by glucagon, a hormone made by the α cells of the pancreas.3,6

Most glucose is stored in the form of lipids, and adipocytes are major sites of insulin action.3-6 Insulin is necessary for the activation of lipoprotein lipase, which breaks down triglycerides into free fatty acids, to be used for energy.6 In individuals with insufficient insulin, such as those with diabetes mellitus, the triglyceride-laden lipoproteins accumulate in circulation. Within muscle tissue, insulin not only allows glucose to be transported into the cells, but it also stimulates amino acid uptake by skeletal muscle and facilitates the use of amino acids in building proteins.6

Furthermore, insulin opposes glucagon, which plays a role in the production of ketone bodies. When insulin levels are low, lipolysis is accelerated, and an overproduction of free fatty acids occurs.5 If glucagon levels are elevated at the same time, the liver produces ketone bodies. Ketone bodies inhibit glucose and fatty acid oxidation, resulting in the use of the ketone bodies for energy. Without insulin, ketoadiposis eventually develops.5,6

Approximately two-thirds of the insulin response to glucose ingestion is a result of the potentiating effect of gut-derived incretin hormones.6 The two main hormones involved in this reaction are glucose-dependent insulinotropic polypeptide (GIP) and glucagon-like peptide-1 (GLP-1).6 In patients with T2DM, the incretin effect produced by these hormones is lost or greatly impaired.3,6

Glucose-dependent insulinotropic polypeptide is produced in the small intestine by K cells in response to carbohydrates and lipids.5 It acts on pancreatic β cells to cause an increase in the release of insulin-containing granules. Glucagon-like peptide-1 is an endogenous molecule secreted by “L” cells in the lumen of the gut (ie, ileum and colon) in response to nutrients. It binds to receptors on pancreatic β cells, stimulating both the synthesis and the secretion of insulin. The GLP-1 molecule is among the most potent insulin-releasing substances known, making it an attractive target of antidiabetes drugs (See chart pages 8-9). It promotes a feeling of satiety and appetite suppression after a meal—effects that are replicated by the incretin mimetics (See chart pages 8-9)—as a result of a decrease of gastric emptying and stimulation of the satiety center in the brain. The incretin mimetics help to supplement natural GLP-1 levels and remain active for a longer period than do the natural molecules.

The GLP-1 and GIP molecules are broken down by dipeptidyl-peptidase-4 (DPP-4), a nonspecific enzyme that also breaks down peptide YY, neuropeptide Y, and growth hormone-releasing hormone. Dipeptidyl-peptidase-4 is also involved in T-cell activation and is expressed on lymphocytes as CD26. Inhibitors of DPP-4 act by preventing the breakdown of GLP-1 and GIP, thereby
Review of guidelines

There are two standard algorithms used for making decisions regarding drug treatment for patients with T2DM. One algorithm is produced by the ADA, 13 and the other by the American Association of Clinical Endocrinologists/American College of Endocrinology (AACE/ACE).15

According to the ADA algorithm, 13 treatment of all patients at diagnosis is initiated with metformin. Treatment then progresses to dual therapy with a sulfonylurea or thiazolidinedione (eg, pioglitazone) or to addition of basal insulin, depending on HbA1c level and patient response. Although effective, this algorithm requires a minimum of 3 to 6 months before a patient might move to dual therapy or insulin and is contingent on the patient's full adherence to prescribed medications and instructions, as well as thorough follow-up with the physician.

Another shortcoming of the ADA algorithm is lack of consideration of the patient's baseline HbA1c level. The ADA algorithm initiates all patients with metformin, regardless of the magnitude of decrease needed to achieve HbA1c goals. The baseline HbA1c level is important given that oral agents decrease HbA1c between 0.5% and 2%, and injectable incretin mimetics decrease HbA1c between 1% and 1.5% as single agents.14 The later addition of another agent generally does not produce a fully additive effect, and a point of diminishing returns becomes apparent.14 Therefore, if the ADA algorithm is used with a patient who has an HbA1c level of 12% at diagnosis, metformin at its maximum dose would bring the HbA1c down to only about 10%—a level that is not acceptable for meeting the ADA's standard HbA1c goal of less than 7%. By contrast, insulin can decrease HbA1c to goal values irrespective of the patient's HbA1c levels at baseline.

The AACE/ACE algorithm 15 uses the patient's baseline HbA1c level as the focus for treatment decisions. For a patient with an HbA1c level of 6.5% to 7.5%, metformin monotherapy is primarily recommended, but the algorithm allows for other oral hypoglycemics or incretin mimetics. If a patient presents with an HbA1c level of 7.6% to 9.0%, dual therapy is initiated with metformin in combination with any of the other oral medications or incretin mimetics. At an HbA1c level of more than 9%, insulin therapy is initiated, either as monotherapy or in conjunction with other agents.14

By basing treatment on the AACE/ACE algorithm 15 for patients presenting with high HbA1c levels, earlier insulin initiation leads to a more rapid breaking of glucose toxicity, allowing for the diminishing of insulin resistance. This development, in turn, leads to lower HbA1c levels and improved quality of life for the patient.16,17 Oral agents are important parts of the arsenal for treating patients with T2DM. Metformin is the typical starting medication because of its ability to lower HbA1c by as much as 2%. Metformin—which is well tolerated and inexpensive and the preferred initial therapy in both the ADA and AACE/ACE guidelines—is only one of the oral options available. Other oral agents most commonly considered would be sulfonylureas, thiazolidinediones and DPP-4 inhibitors.13,15 The injectable incretin mimetics make up an attractive alternative option, because these agents have been shown to decrease HbA1c levels while also promoting weight reduction in the majority of patients.18 An intangible effect of incretin mimetics is that these agents introduce injection therapy into treatment without the stigma that can be attached to insulin.19

Initiation of dual-drug therapy has demonstrated larger HbA1c reductions in drug-naïve patients compared to monotherapy, but dual therapy requires the tradeoffs of using more medicine and risking potentially increased adverse effects.19,20 What remains unclear is whether patients' long-term morbidity and mortality will be improved with initial dual therapy vs initial monotherapy. To clarify this uncertainty, large, prospective, mortality outcome based studies are needed. Until results from such studies become available, physicians should assume that fixed-dose dual therapy can lead to increased patient adherence because of ease of use and overall decreases in cost.21

Nonpharmacologic aspects of care

Regardless of the agent or agents selected, all drug therapies should be initiated along with education of the patient about lifestyle modifications in such areas as nutrition, exercise, and smoking cessation. Weight-loss counseling must be an element of this education. Numerous, rigorously conduct ed, prospective clinical trials have shown that strict adherence to diet and exercise can lower a patient's HbA1c level by at least as much as metformin can.22

Specific to the Hispanic population, data have emerged regarding dietary patterns that may be of use to physicians constructing comprehensive management plans. Young Mexican Americans (aged 41 years or younger) were found to have a diet pattern not statistically different from that of non-Hispanic whites, regardless of whether the Mexican American was born in Mexico or the United States or whether he or she had resided in the United States for less or more than 15 years.23 In ethnic comparisons of prediabetes in overweight and obese children, Hispanic children...
were found to have greater rates of metabolic syndrome than non-Hispanic children (Hispanics, 58%; non-Hispanic whites, 31%; African Americans, 10%), as well as higher levels of triglycerides and fasting blood glucose, lower levels of high-density lipoprotein, and greater waist circumferences.24

Crucial to planning lifestyle modification is the finding that weight loss was the only lifestyle correlate to improving cardiovascular risk in Hispanic children.24 Increasing

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### Comparison of Drug Therapy

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<th>Drug Class</th>
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| Pancreas     | Sulfonylurea - Glimepiride - Glipizide - Glyburide | Continuously stimulates beta cells of pancreas, leading to increased insulin secretion. | - Weight gain and hypoglycemia are most common adverse effects.  
- Glyburide is not optimal for elderly patients, because it is metabolized into active metabolites that can accumulate, causing prolonged hypoglycemia.  
- Contraindicated in patients with T1DM, pregnancy, sulfa hypersensitivity, or renal failure. |
| Meglitinide   | - Nateglinide - Repaglinide | - Stimulates glucose-dependent insulin secretion.  
- Short duration of action. | - To be taken before meal, typically counseled with first bite of meal.  
- If meal is skipped, dose should be skipped.  
- Requires 3-times or 4-times daily dosing.  
- Drug interactions with repaglinide and gemfibrozil.  
- More than doubles half-life of repaglinide, resulting in prolonged hypoglycemia. |
| Amylin analog | - Pramlintide | - Replaces or augments natural amylin released from B cells.  
- Reduces blood glucose by:  
  - slowing gastric emptying, leading to slowed glucose absorption;  
  - stimulating satiety center of brain;  
  - signaling decreased hepatic glucose production. | - Only agent other than insulin that can be used in both T1DM and T2DM.  
- Injectable medication.  
- Can produce weight loss from increased stimulation of brain’s satiety center and adverse effect of nausea with large portions (caused by slowed gastric motility). |
| Basal insulin | - Detemir - Glargine - NPH | Replaces or augments basal (ie, background) insulin continuously throughout day. | - “Nonpeaking” insulin analogs, such as detemir and glargine, have decreased hypoglycemia risk.  
- At small doses, detemir has more consistent coverage with twice-daily dosing; as dose increases, once-daily dosing can provide coverage.  
- Glargine dosing is designed as once daily, but with larger doses, it is beneficial to split dose into once every 12 hours. |
| Prandial insulin | - Aspart - Glulisine - Lispro - Regular | Replaces or augments glucose-dependent insulin. | - Because of rapid onset of action of insulin analogs, mealtime dosing can be at end of meal; dose can then be calculated based on what was actually eaten.  
- Dosing of regular insulin must be 30 minutes before meal because of delayed onset of action (1-2 hours postdose). |
| Liver        | Biguanide - Metformin | - Reduces hepatic glucose output.  
- Increases uptake of glucose in periphery of body. | - Dose titration from 500 mg once daily to 1000 mg twice daily over 4 to 6 weeks reduces gastrointestinal adverse effects.  
- Gastrointestinal adverse effects can promote weight loss, but drug is generally weight neutral.  
- Extended-release formulations can decrease adverse effects when used twice daily.  
- Lactic acidosis is rare, but must be considered with decreased renal function, heart failure, or when patient is hospitalized for surgery or undergoing diagnostic tests with contrast. |
physical activity made no difference in cardiovascular risk of the studied children. In regard to diet as compared to non-Hispanic children, Hispanic children were not found to have increased sugar intake (a correlate of diets high in carbohydrates). Therefore, explanations for the poor cardiometabolic profile of Hispanic children may lie elsewhere. A recent finding regarding Hispanics’ approaches to dieting may prove helpful for diabetes management plans that incorporate weight loss. Compared to other

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<td>Adipose Tissue/Muscle</td>
<td>Thiazolidinedione - Pioglitazone - Rosiglitazone*</td>
<td>- Reduces insulin resistance by binding and activating PPAR-gamma. - Improves insulin sensitivity in skeletal muscle and adipose tissue; secondarily decreases hepatic glucose output.</td>
<td>- Increases HDL-C; pioglitazone decreases triglycerides. - Promotes “lighter, fluffier” LDL-C molecules. - Maximum glucose-lowering effects may take as long as 12 weeks. - Precautions for patients with edema or heart failure and for some premenopausal anovulatory women with potential for resumption of ovulation. - Contraindicated in patients with heart failure of NYHA class III or IV, or with AST/ALT greater than 3X ULN at baseline.</td>
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<td>GLP-1 receptor agonist - Exenatide - Liraglutide</td>
<td>Supplements natural GLP-1 levels, leading to: • glucose-dependent insulin release; • slowing of stomach emptying; • stimulation of satiety center in brain.</td>
<td>- Weight loss is adverse effect, most likely because of portion-size reductions that patients need to make to limit nausea. - Nausea associated with stimulation of nausea center is decreased when injected further away from meal; nausea associated with overconsumption is decreased when injected closer to meal. - Not FDA-approved for use with insulin.</td>
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<td>DPP-4 inhibitor - Linagliptin - Saxagliptin - Sitagliptin</td>
<td>Increases blood concentration of GLP-1 by inhibiting its degradation by DPP-4.</td>
<td>- Weight neutral - No hyperglycemia unless combined with secretagogue or insulin. - Uncommon adverse effects of runny nose and upper respiratory infections, most likely caused by non-selectivity of DPP-4 inhibition.</td>
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<td>Alpha-glucosidase inhibitor - Acarbose - Miglitol</td>
<td>Reduces intestinal glucose absorption by inhibiting pancreatic alpha-amylase and intestinal alpha-glucosidase during carbohydrate digestion.</td>
<td>- Major adverse effects involve gastrointestinal symptoms, with notable cramping and flatulence. - Slow-dose titration can minimize adverse effects, but titration could take 4-6 weeks. - Might be good choice for patients with prediabetes and postprandial hyperglycemia. - Contraindicated in patients with any chronic gastrointestinal disorder that could deteriorate as result of gas formation in intestines.</td>
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<td>Dopamine agonist - Bromocriptine mesylate</td>
<td>Unknown; theorized to help regulate circadian rhythm and hormone levels (eg, cortisol).</td>
<td>- Administered as single dose in morning. - Most common adverse effects are nausea, vomiting, diarrhea, dizziness, and headache. - Can cause orthostatic hypotension. - Probably best used as prediabetes or early diabetes therapy.</td>
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ethnic groups, Hispanics attempted to diet with equivalent frequency (e.g., >65% of Hispanic women), but instead of reducing both caloric and fat intake, Hispanics tended to reduce fat intake without caloric reductions. The offsetting caloric source consisted of carbohydrates—leading to more glucose spikes and pancreatic assaults than experienced by individuals using other dietary mechanisms. This propensity to reduce fats and not address carbohydrates in a population at risk for accumulation of triglycerides and liver fats may require special emphasis on dietary approaches as part of lifestyle modification.

As previously mentioned, weight loss must be a fundamental element of management to maximize improvement in patients with diabetes mellitus. Because a loss of sympathetic response to glucose ingestion is typical in patients with diabetes mellitus, researchers measured changes in sympathetic responsiveness in such patients while a weight-loss program was being implemented. The findings were encouraging in that improvements in sympathetic responsiveness (and correspondingly lower postprandial glucose spikes) were seen with weight loss in individuals diagnosed as having insulin resistance. A further evaluation examined the possible role of physical activity in improving sympathetic responsiveness. Although no additional improvement in sympathetic responsiveness beyond that achieved through weight loss was seen with physical activity, combining physical activity with weight loss did not worsen responsiveness, and the combination resulted in a nearly 20% improvement in fitness.

Accordingly, both the ADA and AACE/ACE guidelines incorporate lifestyle modification in conjunction with initiation of medications. However, lifestyle modifications are difficult to institute and perhaps even more difficult to maintain. Therefore, to be successful, diabetes management teams must include at least one member who is continually addressing lifestyle modification with the patient.

**Maximizing efficacy of care**

There are many avenues that can be explored to help patients with not only lifestyle modification, but also with monitoring of drug regimens. A complete diabetes education course would be optimal for all patients, but such courses can be cost-prohibitive and difficult to attend because of work schedules. Furthermore, in many communities, such courses are given only in English and have long waiting lists. Another problem with a diabetes education course is that it provides only a “one-shot” approach to education, without the continual reinforcement that is required for optimal management.

 Pharmacists in many communities can provide additional education and monitoring of medication use for patients with diabetes mellitus. Most patients who have this condition are at the pharmacy at least once a month, which may be 3 to 6 times more often than they are at their physician’s office. The pharmacist can provide timely advice to the patient and—through bilateral communication with the patient’s physician (as well as in compliance with all state and federal statutes)—make any adjustments to treatment that might be deemed appropriate.

As an example of this symbiotic relationship, a patient is started on metformin, 500 mg daily, and scheduled to return for follow-up in three months to evaluate his HbA1c response. As part of a protocol-driven medication therapy management (MTM) partnership involving the patient’s physician and a local community pharmacist, the patient could have the metformin dose incrementally increased by the pharmacist to optimal dosing (1000 mg twice daily) during the three-month period as he comes in for routine refills. A bonus aspect of this partnership is the adherence monitoring that takes place simultaneously with adverse-effect monitoring. As a consequence of the MTM partnership, at the three-month physician follow-up the patient is at optimal metformin dose, and medication efficacy can be more efficiently and accurately assessed by the time-taxied physician.
If the intervening pharmacy visits were cut from this scenario, metformin doses would have to be increased at each of the next three-month follow-up appointments with the physician—delaying the achievement of the optimal dose until nine months after therapy initiation. One of the authors (S.B.N.) has found that the MTM partnership improved patient outcomes and decreased inappropriate medication use in a rural population in northwestern Indiana.

Although the described MTM scenario is only an anecdotal example, the medical literature contains easily retrieved outcome-based studies demonstrating that physician-pharmacist teams can lead to not only improved glycemic control, but also reduced healthcare costs as a result of decreased hospitalizations and reduced long-term complications.26-34 An intangible aspect of this team approach is the additional contact between patient and healthcare professional in a nonthreatening environment (ie, a community pharmacy). This aspect was found to be beneficial in building relationships between the patient and the healthcare team. This relationship is an essential component in positive therapeutic outcomes within the Hispanic community.32

The CDC has recognized team approaches as well. In recent CDC-sponsored publications—“Guiding Principles for Diabetes Care: For Health Care Professionals” and “Redesigning the Health Care Team: Diabetes Prevention and Lifelong Management”—team approaches to managing diabetes mellitus were called “essential.” (These publications are available on the National Diabetes Education Program website at http://ndep.nih.gov.)

To optimize outcomes in patient care, cultural awareness is just as important as team involvement. Team members need to learn to treat patients within the context of the patients’ cultures. For example, starting a conversation with a Hispanic patient by asking, “What is the most difficult thing for you about having diabetes?” may prove more impactful in outcomes than any drug prescribed.

There are many cultural and societal issues that need to be addressed to maximize efficacy of diabetes management in Hispanic patients. However, a complete and accurate presentation of these issues exceeds the scope of this article. Following are a few selected key aspects that we hope may be beneficial to physician-led clinical teams.

Cultural barriers to optimizing care for Hispanic patients with diabetes mellitus may best be summarized as two main issues: resistance to seeking care and the stigma of insulin. Resistance to seeking medical care may be compounded by an inability to access care as a result of economic, financial or language barriers—problems that are well established in Hispanics, especially among men.33-37 Consequently, by the time T2DM is diagnosed in a Hispanic patient, the disease is typically in an advanced stage, including the beginning of complications. This issue is further complicated by the previously discussed evidence that Hispanics present with a disproportionately large amount of renal disease compared to non-Hispanic matched controls.2 A delay in early management in a patient who is potentially predisposed to a rapid course of disease is not a good combination.

A difference in perception between Hispanics and non-Hispanics regarding the benefit or utility of insulin has been documented.37 It has been postulated that many Hispanics view insulin as the therapy of last resort or as being responsible for causing the complications or demise of a loved one.28-30 Because of this mindset, the physician may need to spend greater time convincing the Hispanic patient of the value and necessity of insulin as a therapeutic modality—and during this time, the patient’s diabetes may continue to progress outside acceptable parameters.

Overcoming the stigma of insulin therapy can be a challenge. It is not just the Hispanic culture that incites this stigma. For too long, healthcare professionals used insulin as a threat to improve patients’ “bad behavior” with respect to lifestyle changes and medication adherence. A more appropriate approach would be to initiate conversations about insulin at the time of diagnosis, but not as a threat or “nuclear option.” It should be made clear to the patient that insulin is an important aspect of treatment for diabetes mellitus—and arguably the most effective treatment option at managing blood glucose levels with the least amount of adverse effects.40 Concurrent with the introduction of insulin into the conversation at the time of diagnosis, the concept of disease progression is paramount. Regardless of how well a patient may adhere to lifestyle modifications and medication regimens, there may come a point in time at which insulin is needed to slow the course of disease.

Final notes

Many options exist to optimize treatment for Hispanics who are at risk for, or who have, diabetes mellitus. Although certain early interventions in individuals with diabetes have shown promise, aggressive management and engagement of the patient’s entire support system—especially in early identification of at-risk pediatric and adolescent Hispanics—are likely to be crucial to successful outcomes. The physician is strongly encouraged to engage pharmacists and other nonphysicians in the community who have skill sets complementary to achieving desired results in patients.

References


