



### Inside Bangladesh

#### Intervention, recruitment and evaluation challenges in the Bangladeshi community

##### Background:

The prevalence of type 2 diabetes varies with ethnic origin. In the UK, people from the Indian subcontinent are particularly susceptible to diabetes and one in five older South Asians have diabetes. The adoption of self-management skills is necessary for people to manage their diabetes. However, people from South Asian communities generally know less about their diabetes and its management compared to Caucasian patients. The incidence of type 2 diabetes is increasing worldwide and diabetes is four times more common among ethnic minority groups than among the general Caucasian population. This study reflects on the specific issues of engaging people and evaluating interventions through written questionnaires within older ethnic minority groups.

##### Methods:

The original protocol set out to evaluate an adapted version of the X-PERT<sup>®</sup> patient program using questionnaires and interviews. Results: Questionnaires, even verbally completed, were unsuccessful and difficult to administer as participants found the questionnaire structure and design difficult to follow and did not perceive any benefit to completing the questionnaires. The benefits of attending the course were also poorly understood by participants and in many cases people participated in coming to the course as a favour to the researcher. Engaging participants required word of mouth and the involvement of active members of the community.

##### Conclusion:

The use of written methods of evaluation may not be appropriate in older ethnic minority groups and interventions aimed at older ethnic minority groups should be designed with their specific needs in mind. Evaluation of interventions aimed at the Bangladeshi community need to be carefully planned and targeted to be relevant to this specific community. These findings may also be appropriate for other hard to reach communities.

Source: SM Choudhury, S Brophy, MA Fareedi, B Zaman, P Ahmed and DRR Williams. BMC Medical Research Methodology 2008, 8:64.



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### Nocturnal Hypoglycemia Common in Type 1 Diabetes

Prolonged nocturnal hypoglycemia is common in type 1 diabetics. This study shows the value of continuous glucose monitoring in identifying hypoglycemia that occurs overnight for prolonged periods of time while the patient with type 1 diabetes is sleeping. Severe prolonged hypoglycemia can lead to seizures and coma.

Dr. Beck of the Jaeb Center for Health Research, Tampa, Florida, and colleagues studied 176 children and adults, ages 8 to 72, with type 1 diabetes. The researchers had continuous glucose monitoring data for 36,467 nights (with a minimum of 4 hours of monitoring per night).

They defined prolonged hypoglycemia as at least two consecutive readings no higher than 60 mg/dL within a 20 minute period.

These events occurred on an average of 8.5% of nights, the authors found. The median percentage of nights with prolonged hypoglycemia, per patient, was 7.4% (or about twice per month, the authors said). Only 3 patients had no hypoglycemic nights.

In 23% of hypoglycemic nights, these events lasted for at least 2 hours.

Factors significantly associated with nocturnal hypoglycemia were lower baseline HbA1c levels and nocturnal hypoglycemia during baseline blinded continuous glucose monitoring.

"Identifying patients in whom nocturnal hypoglycemia is occurring frequently is important so that insulin dosing and bedtime snacks can be adjusted to try to prevent its occurrence.

*Diabetes Care 2010, March 3rd online paper.*

# Diabetescope

### Dexamethasone Implants Improve Diabetic Macular Edema

An intravitreal dexamethasone delivery system improves diabetic macular edema. The intravitreal implant (Ozurdex, Allergan) contains dexamethasone in a solid polymer drug delivery system. Literature from its manufacturer says that in earlier clinical trials, 20% to 30% of patients had at least a 15-letter (3 lines) improvement in best-corrected visual acuity within two months after implantation, with improvements lasting approximately one to three months after onset of this effect.

In the current study, Dr. Julia A. Haller of the Wills Eye Institute, Philadelphia, and colleagues focused on 171 clinical trial subjects with persistent diabetic macular edema (one eye in each person). The phase II trial had three arms: a 250 ug dexamethasone implant, a 700 ug system, or observation only.

At 90 days, 33.3% of the 700 ug group, 21.1% of the 250 ug group, and 12.3% of controls had improvements of at least 10 letters in their best-corrected visual acuity.

The difference between the higher dose implant and observation was statistically significant at days 60 and 90. At 180 days, the visual acuity improvement persisted in 30% of the 700 ug group, 19% of the 350 ug group, and 23% of the observation group; at this point, the differences were no longer significant.

Both treatment groups had significant improvements in central retinal thickness and fluorescein leakage compared with the observation group.

The implants were well tolerated and they appear "to be a promising new treatment option for eyes with persistent diabetic macular edema.

*Arch Ophthalmol 2010;128:289-296.*



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## Early and Aggressive Initiation of Insulin Therapy for Type 2 Diabetes: What Is the Evidence?

Type 2 diabetes mellitus is a progressive disease in which  $\beta$ -cell function continually declines and eventually fails, ultimately requiring nearly all patients to be placed on insulin therapy. An increasing body of evidence suggests that early intensive glycemic control reduces long term vascular outcomes and potentially may prolong  $\beta$ -cell lifespan and function. Ultimately, most patients will require insulin therapy, although insulin is still all too often thought of as "last resort" or "end-stage" therapy. This and other misperceptions frequently limit the early initiation of insulin therapy, even among patients for whom oral agents are no longer adequate.

A variety of insulin analogs are now available that lower the risk of hypoglycemia and result in less weight gain, thus providing the tools to overcome barriers commonly associated with insulin therapy. New insulin analogs more closely mimic the kinetic profile of endogenous insulin and allow for flexible dosing in pen devices that are generally well received by patients. Clinical outcome data, together with the safety and convenience of insulin analogs and newer insulin-delivery devices, may make early initiation of insulin therapy more attractive. The objective of this review is to present recent clinical evidence in favor of early and aggressive bloodglucose lowering in patients with type 2 diabetes, and, in this context, to discuss and highlight real-world clinical experiences for type 2 diabetes disease management.

### Early and Aggressive Intervention Reduces Long-Term Vascular Risk

Cardiovascular disease is the major cause of morbidity and mortality in patients with diabetes. Thus, glycemic control is the primary therapeutic goal in the management of type 2 diabetes. Three laboratory measures are recommended to gauge the level of glycemic control attained by individual patients. A1C values reflect glycemic exposure during a period of ~ 3 months. The expected A1C value for people with normal glucose metabolism is 4.0-6.0%. Recommended target values for individuals with diabetes are < 7% or <6.5%. Individualized targets based on patients' entire clinical situation are also important. Fasting plasma glucose (FPG) and postprandial plasma glucose (PPG) values provide snapshots of basal glucose metabolism (i.e., hepatic glucose production) and, most importantly, exposure to postprandial glucose excursions, which have recently been linked to overall vascular damage. Tight glycemic control is crucial for reducing the incidence of retinopathy, nephropathy, and neuropathy in patients with diabetes, and evidence suggests that early control prevents macrovascular events many years down the road (i.e., induces a "metabolic memory"). Results from

Table 1. Goals for Glycemic Control

	ADA	AACE
A1C (%)	<7	$\leq 6.5$
FPG (mg/dl)	70-130	<110
PPG (mg/dl)	<180	<140

ADA, American Diabetes Association; AACE, American Association of Clinical Endocrinologists.

the U.K. Prospective Diabetes Study (UKPDS) showed that aggressive glycemic control, with sulfonylureas or insulin in patients newly diagnosed with type 2 diabetes significantly reduced the risk of any microvascular endpoint compared to conventional treatment. There was no significant difference in macrovascular risk among those treated intensively with chlorpropamide, glibenclamide, or insulin during the 10-year study period. Reduction in risk of myocardial infarction was of borderline significance with intensive glycemic control. No significant benefit was seen for other macrovascular endpoints. The results of a 10-year poststudy follow-up revealed the long-term benefit of early glycemic control in the UKPDS population. At study end, the median A1C value was 7% in the intensive treatment group and 7.9% in the conventional treatment group, although values increased steadily during the 10-year study period. After study end, the course of therapy was left to the discretion of the patients and their physicians, and differences in glycemic control had disappeared between treatment groups at the end of the first poststudy year. Glycemic control was similar during this follow-up period, and the A1C values were statistically similar across treatment groups during the course of the poststudy follow-up. Intriguingly, those who had received intensive treatment soon after diagnosis had significantly lower rates of microvascular disease. In addition, these patients had lower rates of any diabetes related endpoint, diabetes-related death and death from any cause. A statistically significant reduction of risk for myocardial infarction was observed in patients who had been in the intensive treatment groups early but who had similar glycemic control in the follow-up period. Results were further analyzed for the subgroup of overweight subjects at study entry and those who were treated with metformin rather than a sulfonylurea. During the poststudy follow-up, patients in the metformin group also initially showed lower risk of any diabetes endpoint, diabetes related death, death from any cause, and myocardial infarction. Similar findings have been observed in the landmark Diabetes Control and Complications Trial (DCCT), in which intensive control prevented microvascular complications, despite the fact that glycemic control of the intensive group rapidly decayed to that of the "standard"

## Initiation of Insulin Therapy for Type 2 Diabetes

therapy group at the end of the study. In the followup Epidemiology of Diabetes Interventions and Complications (EDIC) study, a similar large effect of early glycemic control on cardiovascular events was noted. Both the UKPDS and the DCCT/EDIC studies provide a strong rationale that early aggressive intervention in diabetes will dramatically lessen the burden of cardiovascular disease many years later.

### Rationale for Early Initiation of Insulin Therapy

The fundamental scientific and clinical question of whether the progressive nature of diabetes can be modified remains of great interest. In proof of principle, the Diabetes Prevention Program demonstrated that an intensive lifestyle intervention was most effective at reducing progression to diabetes in high-risk individuals, followed by metformin therapy. There has been similar interest in understanding whether early intervention with insulin may be fundamentally disease-altering, potentially by protecting  $\beta$ -cell function. A recent, randomized, parallel-group study of 382 patients with newly diagnosed type 2 diabetes provides intriguing support to this hypothesis. The effects of intensive, short-term insulin therapy on  $\beta$ -cell function was evaluated in this trial, in which patients were randomly assigned to treatment with continuous subcutaneous insulin therapy, multiple daily insulin injections, or oral hypoglycemic agents. Once patients achieved and sustained ontherapy normoglycemia for 2 weeks, pharmacological treatment was stopped. Normoglycemia was attained by > 95% of patients in the insulin treatment groups compared to 84% of those receiving oral agents. Glycemic control was reached significantly faster with insulin, and at 1 year after treatment, 51% of those who had received continuous insulin and 45% of those who had received multiple daily insulin injections remained normoglycemic compared with 27% of patients randomized to the oral treatment group.  $\beta$ -Cell function was measured at the end of therapy and after 1 year using homeostasis

model assessment of basal  $\beta$ -cell function (HOMA B) and acute insulin response. Patients treated with continuous insulin therapy had an increase in HOMA B of 160% compared to 105% for those treated with oral agents, an impressive, if not somewhat surprising, finding. Another smaller study (n = 20) found an immediate improvement in  $\beta$ -cell function after switching patients from sulfonylurea to preprandial rapid-acting insulin analog therapy. Finally, a 4-year randomized study of 49 patients who had been diagnosed with type 2 diabetes within 2 years of study entry compared the effects of insulin and glibenclamide on  $\beta$ -cell function, metabolic control, and quality of life. Because glibenclamide stimulates endogenous insulin secretion, the study was designed to assess whether such stimulation accelerates  $\beta$ -cell failure. During the first year of treatment, A1C values were similar in the two treatment groups.

Table 2. Summary of OAD Interventions as Monotherapy			
Intervention	Expected Decrease in A1C (%)	Advantages	Disadvantages
<b>Tier 1: Well-Validated Core</b>			
<b>Step 1: Initial therapy</b>			
Lifestyle changes to decrease weight and increase activity	1–2	Broad benefits	Insufficient for most in first year
Metformin	1–2	Weight neutral	Gastrointestinal side effects, contraindicated with renal insufficiency
<b>Step 2: Additional therapy</b>			
Insulin	1.5–3.5	No dose limit, rapidly effective, improved lipid profile	1–4 injections daily, monitoring, weight gain, hypoglycemia, high cost of analog insulin products
Sulfonylurea	1–2	Rapidly effective	Weight gain, hypoglycemia (especially with glibenclamide or chlorpropamide)
<b>Tier 2: Less Well Validated</b>			
Thiazolidinedione	0.5–1.4	Improved lipid profile and potential decrease in myocardial infarction (with pioglitazone)	Fluid retention, congestive heart failure, weight gain, bone fractures, high cost, potential increase in myocardial infarction (with rosiglitazone)
GLP-1 agonist	0.5–1.0	Weight loss	Two injections daily, frequent gastrointestinal side effects, long-term safety not established, high cost
<b>Other Therapy</b>			
$\alpha$ -Glucosidase inhibitor	0.5–0.8	Weight neutral	Frequent gastrointestinal side effects, thrice-daily dosing, high cost
Glinide	0.5–1.5*	Rapidly effective	Weight gain, thrice-daily dosing, hypoglycemia, high cost
Pramlintide	0.5–1.0	Weight loss	Three injections daily, frequent gastrointestinal side effects, long-term safety not established, high cost
DPP-4 inhibitor	0.5–0.8	Weight neutral	Long-term safety not established, high cost

**Table 3. Onset, peak, and duration of insulin actions**

Insulin*	Onset	Peak	Effective Duration
Rapid-acting • Insulin aspart • Insulin lispro • Insulin glulisine	5–15 minutes	30–90 minutes	< 5 hours
Short-acting • Regular insulin	30–60 minutes	2–3 hours	5–8 hours
Intermediate (basal) • NPH	2–4 hours	4–10 hours	10–16 hours
Long-acting (basal) • Insulin glargine • Insulin detemir	Not applicable	Relatively flat	Up to 24 hours
Premixed • 75% lispro protamine/25% lispro • 70% aspart protamine/30% aspart • 70% NPH/30% regular	5–15 minutes 5–15 minutes 30–60 minutes	Dual Dual Dual	10–16 hours 10–16 hours 10–16 hours

\*Assumes 0.1–0.2 units/kg/injection. Onset and duration may vary by injection site.

During this progression, loss of glucose control with oral agents results in glucose toxicity and worsening pathophysiology. In experimental models, prolonged exposure to hyperglycemia has been shown to result in glucotoxicity and oxidative stress, culminating in  $\beta$ -cell destruction and microvascular and macrovascular complications. The timely addition of insulin to oral agents can prevent this cycle of disease progression and eliminate the “stuttering” pattern of loss of glycemic control. Insulin is

However, during the next 3 years, glycemic control deteriorated faster in the glibenclamide group, and at year 4, A1C values were significantly higher than in the insulin treatment group. Fasting insulin levels after acute treatment withdrawal were significantly higher in the insulin group throughout the study, suggesting that patients receiving insulin retained greater capacity for  $\beta$ -cell response and supporting the hypothesis that stimulation of endogenous insulin production may contribute to  $\beta$ -cell failure. Of note, both treatments were well tolerated, and no significant effects on quality of life were measured between the two groups. Taken together, the results of these studies are consistent and suggest that early insulin supplementation may alter the progressive course of diabetes. This may be due to protection of, and possibly restoration of,  $\beta$ -cell function. More studies will clearly be required to verify and extend these findings and to understand specific biological mechanisms involved.

### Initiating Insulin Therapy

A range of pharmacotherapies other than insulin are available to meet the glucose-lowering needs of patients across the spectrum of type 2 diabetes progression. Oral antidiabetic drugs (OADs) are often used as initial therapy. Because type 2 diabetes is a progressive disease,  $\beta$ -cell mass and function gradually decrease to the point at which A1C levels rise despite the use of more than one OAD.

clearly the most effective way to control blood glucose, but it also presents many therapeutic barriers for physicians and patients alike. As shown in Table 3, insulins with different pharmacodynamic profiles are available, allowing for three possible strategies to initiate insulin therapy: 1) basal insulin, 2) basal/ bolus insulin, or 3) premixed insulin. Typically, the first strategy to consider is the early addition of a basal insulin to an OAD regimen. The long-acting basal insulin analogs insulin detemir and insulin glargine have a highly favorable pharmacodynamic profile (a long, relatively flat insulin time-action curve lasting up to 24 hours) that attenuates the risk of hypoglycemia. Compared to neutral protamine Hagedorn (NPH) insulin, both detemir and glargine have demonstrated comparable efficacy for glycemic control, a potential for once-daily dosing, and less hypoglycemia, and, with insulin detemir, a propensity toward less weight gain. Despite having fundamentally different pharmacological properties at the insulin molecule level, clinical trial data have shown that insulin detemir and insulin glargine have similar glycemic efficacy, but somewhat different effects with respect to weight gain. Insulin detemir has consistently shown effective glycemic control accompanied by either weight loss or lower rates of weight gain compared to human insulins. Most patients will ultimately require prandial insulin in addition to basal insulin as  $\beta$ -cell function declines. Because diabetes is a

heterogeneous disorder, some patients may require intensive basal/bolus therapy earlier than others. Basal/bolus therapy using rapid-acting insulin at mealtimes in addition to a basal insulin analog is highly effective and allows flexibility in both the timing and amount of prandial insulin dosing. Indeed, this type of regimen is considered state-of-the-art and is clearly ideal for many patients with diabetes. Premixed insulins may provide an easier means for achieving near-normal insulin profiles, but they provide less flexibility. Premixed biphasic insulins provide both a basal and prandial insulin component in a single injection and can be administered once or twice daily as initial therapy for type 2 diabetes. In some patients whose hyperglycemia is not adequately managed with oral agents, starting with biphasic insulin to provide basal and prandial insulin can be as effective as basal insulin plus metformin. For example, the INITIATE study found that twice-daily biphasic insulin aspart 70/30 was more effective than glargine once daily in achieving target A1C levels, but it was associated with greater weight gain and more frequent minor hypoglycemic episodes. The 1-2-3 Study evaluated the efficacy and safety of biphasic insulin aspart 70/30 administered once, twice, or three times daily in patients with type 2 diabetes. In this 48-week observational study, 41% of patients achieved target A1C values of < 7% with once-daily dosing, 70% with twice-daily dosing and 77% with thrice-daily dosing. Although the patients in this study were not necessarily recently diagnosed, the results showed that glycemic control can be achieved with biphasic insulin in patients for whom oral agents are not enough. Intensive patient education regarding initiating insulin therapy, treating hypoglycemia, monitoring blood glucose, and improving diet and lifestyle can alleviate concerns and increase the likelihood of safe and successful treatment.

### Implications for Clinical Practice

The following case studies are designed to illustrate real-world clinical situations in which initiation of insulin therapy may represent a good therapeutic choice.

**Case 1.** John was a 63-year-old African-American office worker with a BMI of 37 kg/m<sup>2</sup>. His diet was high in carbohydrates, fat, and salt, and he got little exercise. His A1C was 11.2%, and his FPG was 280 mg/dl. His initial therapy consisted of metformin 2,000 mg daily, plus a diet and exercise plan. At his 3-month follow-up visit, his A1C was 9.4%, and his FPG was 210 mg/dl. Thus, in this first case, glycemic control was not achieved after 3 months of therapy with metformin and lifestyle changes in a patient newly diagnosed with type 2 diabetes. The initial therapy prescribed for John was the maximum dose of metformin in combination with lifestyle interventions. Metformin is a good choice for an oral agent because it is weight-neutral and inexpensive and can lower A1C values by 1.5%. Weight

loss and dietary changes are crucial to achieving glycemic control and ideally will result in A1C decreases of 1 or 2%. After 3 months, however, John's A1C, although lower, was still well above the target value. One approach to increasing the intensity of his therapy would be to add a second oral agent to the existing regimen in the form of a sulfonylurea. However, a recent meta-analysis has shown that adding a sulfonylurea to metformin is unlikely to reduce A1C by >1%, and deterioration of glycemic control after addition of a sulfonylurea to metformin is frequent within 6 months. Moreover, this approach is associated with weight gain and a higher incidence of hypoglycemic events. Initiating a basal insulin regimen in addition to the second OAD may allow the patient to control his hyperglycemia but will not reduce the effect on his weight. The American Association of Clinical Endocrinologists (AACE) recommends that treatment-naïve individuals whose initial A1C value is >10% be started on insulin therapy. Consistent with these recommendations, John could be started on a long-acting insulin, such as insulin detemir or insulin glargine. Initiation of long-acting insulin might be expected to reduce the A1C level by 2–2.5%, avoiding the addition of a second oral agent. Other alternatives might include an oral dipeptidyl peptidase-4 (DPP-4) inhibitor (i.e., sitagliptin) or a long-acting glucagon-like peptide 1 (GLP-1) analog, although neither would be predicted to be as efficacious in this patient, whose A1C-lowering goal is still nearly 2.5%.

**Case 2.** Nicole was a 17-year-old African-American student with a BMI of 25 kg/m<sup>2</sup>. Upon presentation, her A1C was 8.6%, and her FPG was 231 mg/dl. She had a positive family history of type 2 diabetes and had experienced weight loss and symptoms of polyuria and polydipsia. Thus, in this case, a patient with new-onset diabetes (presumed to be type 2 diabetes) was started on metformin, 500 mg twice daily, and a split-mixed insulin regimen of NPH and lispro, in addition to fluids and given diabetes education. At her 1-month follow-up visit, she was found to be positive for islet cell autoantibodies, GAD antibodies, and ICA-512 antibodies, all of which are diagnostic of type 1 rather than type 2 diabetes. Her A1C was 7.8%. Her metformin was discontinued because of her positive antibody studies, but her insulin regimen was continued. At her 3-month follow-up, Nicole's A1C was 5.9%. With her African-American race and family history of diabetes, combined with the growing prevalence of type 2 diabetes, many physicians would have assumed that she had type 2 diabetes. Fortunately, her physician screened for antibodies against β-cells, clearly identifying her disease as autoimmune-mediated type 1 diabetes. She also had evidence of insulin resistance that may be referred to as "double diabetes." Had the correct diagnosis not been made and had she continued treatment with metformin monotherapy, the disease may have

progressed to diabetic ketoacidosis. The use of other secretagogues would also have been counterproductive and contraindicated. Finally, the insulin regimen chosen reflects thoughtful attention to treating both fasting and postprandial hyperglycemia.

### Conclusions

These clinical case studies exemplify the diversity of patients who may benefit from early insulin initiation. Ultimately, it is hoped that early initiation of therapy will not only prevent short-term complications, but also reduce long-term morbidity and mortality and potentially alter the natural history of the disease. This latter concept is currently of intense interest. Although optimal disease management is patient-specific, achieving and maintaining tight glycemic control are the primary goals of therapy. Because many type 2 diabetic patients will eventually require insulin therapy, overcoming fears and therapeutic barriers to initiating therapy early as needed are essential for reducing the vascular comorbidities of this highly prevalent disease in patients of all ages. Fortunately, a number of new clinical tools are available, including both prandial and basal insulin analogs, new insulin-delivery devices, and an ever-improving knowledge of the pathophysiology and natural history of diabetes.

## Congratulations !

### The Winners of *diabetes* Quiz Competition

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