Original Article

# Clinical experience of switching from glargine or neutral protamine Hagedorn insulin to insulin detemir in type 2 diabetes: Observations from the Indian cohort in the A$_1$chieve study

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## ABSTRACT

**Aim:** To explore the clinical safety and effectiveness of insulin detemir (IDet) in a subgroup of Indian patients with type 2 diabetes (T2D) switched from either insulin glargine (IGlar) or neutral protamine Hagedorn (NPH) insulin in the 24-week, non-interventional A$_1$chieve study. **Materials and Methods**: Indian patients with T2D switching from pre-study IGlaror NPH insulin to IDet were included. Safety and effectiveness outcomes were evaluated by the physicians in local clinical settings. **Results**: A total of 102 patients switched from IGlar to IDet (GLA group) and 39 patients switched from NPH insulin to IDet (NEU group). At baseline, the mean glycated hemoglobin A$_{1c}$ (HbA$_{1c}$) levels were 9.9 ± 1.8% in the GLA group and 9.1 ± 1.2% in the NEU group. No serious adverse drug reactions, serious adverse events, or major hypoglycemic events were reported in either group throughout the study. At baseline and Week 24, 11.8% and 7.5% of patients, respectively, reported overall hypoglycemic events in the GLA group. No hypoglycemic events were reported at Week 24 in the NEU group. At Week 24, the mean HbA$_{1c}$ levels were 7.6 ± 0.9% in the GLA group and 7.3 ± 0.7% in the NEU group. The mean fasting plasma glucose, postprandial plasma glucose and quality of life also appeared to improve over 24 weeks. **Conclusion:** Switching to IDet therapy from IGlar and NPH insulin was well-tolerated and appeared to be associated with improved glycogenic control in Indian patients.

**Key words:** Glargine, India, insulin detemir, neutral protamine Hagedorn insulin, type 2 diabetes

## Introduction

Despite the availability of a large number of therapeutic agents for the management of type 2 diabetes (T2D), patients continue to present with high levels of glycated hemoglobin A$_{1c}$ (HbA$_{1c}$) in actual clinical practice.[1]

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Insulin is the most potent therapy for T2D[2]; however, physicians often take a conservative approach to insulin dose optimization citing lack of familiarity and the risks of hypoglycemia and weight gain as some of the main concerns associated with its use.[3]

It is acknowledged that insulin therapy tailored towards the requirements of individual patients would be more effective in helping patients manage hyperglycemia, while mitigating the risks of hypoglycemia and weight gain. A study by Riddle *et al.*[4] suggests that basal (fasting) glucose, rather than postprandial plasma glucose (PPPG), tends to contribute more towards overall hyperglycemia at higher HbA$_{1c}$ levels in T2D patients. Basal insulin therapy

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could therefore help decrease hyperglycemia in T2D patients by providing additional glycemic control.[5]

Insulin detemir (IDet) is a basal insulin analogue with a protracted duration of action compared to neutral protamine Hagedorn (NPH) insulin. IDet remains soluble in the subcutaneous depot following injection, which may contribute to its lower within-subject variability with regard to NPH insulin and another basal analogue, insulin glargine (IGlar).[6]

Studies have found that IDet provides $HbA_{1c}$ reductions in T2D patients comparable to those provided by IGlar[7,8] and NPH insulin.[9] IDet therapy is also associated with significantly less weight gain compared to IGlar and NPH insulin in T2D management.[10] It has also been noted that glycemic control improved following IDet therapy in T2D patients previously treated with other basal insulins in combination with oral glucose-lowering drugs (OGLDs).[11]

In India, the T2D epidemic is widespread with a reported diabetes prevalence of 9.0% in 2011.[12] By 2030, the diabetes prevalence is set to increase to 10.6%, marking the urgent need to improve T2D management in the country.[12] In developing nations such as India where diabetic complications are common and a large segment of the population is of working age, the high T2D prevalence can have serious economic implications as well.[13]

Evidence from randomized controlled trials serves as the main foundation for treatment decisions in T2D care. However, the patient populations of these trials are selected based on restrictive criteria and may not be truly representative of the patients seen in actual clinical practice. Large observational studies such as A$_1$chieve,[14] on the other hand, can provide relevant and timely data that would help in gauging patient responses to different therapies in a heterogeneous setting. This sub-analysis of the A$_1$chieve study aimed to determine the clinical safety and effectiveness of insulin detemir in Indian patients switched from previous NPH insulin or IGlar therapy.

## Materials and Methods

### Study design

In the open-label, 24-week, non-interventional A$_1$chievestudy, the clinical safety and effectiveness of the Novo Nordisk insulin analogs, IDet (Levemir®), biphasic insulin as part 30 (NovoMix 30®) and insulin as part (NovoRapid®) in the treatment of T2D was evaluated in routine clinical care.[14] Here, the clinical safety and effectiveness of IDet was explored in Indian patients switched from either IGlar or NPH insulin. Patients were recruited between May 2009 and December 2010 from 621 centers across India.

Home *et al.*[14] have described the methods and procedures of the A$_1$chieve study in detail. In brief, the prescription of IDet was determined by the local physicians, who also supervised all aspects of the patients' treatment. Concomitant OGLD use was permitted at the physician's discretion.

There were no special investigations or procedures. The physicians performed all evaluations at routine visits to the local clinics, following which the data were transferred to standard case report forms.

### Selection criteria

Indian patients switching therapy from either pre-study IGlaror NPH insulin to IDet were included in this sub-analysis. Patients were excluded if they had received treatment with any of the study insulins for more than 4 weeks before the start of the study. Pregnant or breastfeeding women were excluded as were those who intended to become pregnant within 6 months from the start of the study. Study approval was obtained from the relevant local authorities and signed informed consent was obtained from all patients.

### Outcomes

The primary outcome measure was the incidence of serious adverse drug reactions (SADRs), including major hypoglycemic events, from baseline to Week 24.

Secondary outcomes included the number of serious adverse events (SAEs) and the changes in the proportion of patients reporting hypoglycemic events in the 4 weeks before baseline and Week 24. Additional outcomes comprised the change from baseline to Week 24 in $HbA_{1c}$, fasting plasma glucose (FPG), PPPG, systolic blood pressure (SBP), body weight, lipids (total cholesterol, triglycerides, high-density lipoprotein [HDL] cholesterol, and low-density lipoprotein [LDL] cholesterol) and quality of life (QoL).

QoL was determined using the EuroQol Visual Analogue Scale (EQ-VAS) that rates an individual's current health state on a scale of 0 (worst score) to 100 (best score).

Local laboratories were used for laboratory measurements and followed local standardization and quality control procedures.

### Statistical analyses

Statistical analyses were performed for Indian patients switching from pre-study IGlar and NPH insulin.

Descriptive statistics (mean, SD) and frequency tables (*n*, %) were used to summarize continuous and discrete variables, respectively.

The change from baseline to Week 24 in the proportion of patients reporting at least one event of hypoglycemia was analyzed using McNemar's test. The change from baseline to Week 24 for $HbA_{1c}$, FPG, PPPG, SBP, body weight, lipids, and QoL was analyzed using a paired *t*-test. Two-sided testing with 5% significance was employed. No *P* values are presented as the number of patients analyzed was less than 100 for all endpoints.

Data were analyzed by Novo Nordisk using SAS (Version 9.1.3).

## Results

### General characteristics

A total of 102 patients switched therapy from pre-study IGlar to IDet (GLA group), while 39 patients switched from pre-study NPH insulin to IDet (NEU group). Demographic and baseline characteristics of the GLA and NEU groups are presented in Table 1.

At baseline, the most commonly used OGLDs were metformin and sulfonylureas in both groups [Table 1].

The three most common reasons reported by physicians for therapy change were to improve glycemic control (for 96.1% of patients), reduce the risk of hypoglycemia (42.2% of patients) and improve weight control (32.4% of patients) in the GLA group. In the NEU group, the most common reasons for therapy change were to improve glycemic control (97.4% of patients), reduce the risk of hypoglycemia (51.3% of patients) and try a new insulin (25.6% of patients).

### Insulin dose and dosing frequency

Insulin dose and dosing frequency details are presented in Table 2.

The mean pre-study IGlar dose was 0.26 ± 0.13 U/kg, and 84.3% of patients followed *qd* dosing. The mean IDet dose was 0.23 ± 0.09 U/kg at baseline and 0.25 ± 0.12 U/kg at Week 24. At baseline, 87.3% of patients dosed IDet *qd*, while at Week 24, 62.7% and 37.3% of patients followed *qd* and *bid* dosing, respectively.

The mean pre-study NPH insulin dose was 0.27 ± 0.16 IU/kg, and 64.1% and 35.9% of patients followed *qd* and *bid* dosing, respectively. At baseline and Week 24, the mean IDet dose was 0.24 ± 0.09 U/kg and 0.28 ± 0.09 U/kg, respectively. While 87.2% of patients followed *qd* dosing at baseline, 67.7% and 32.3% of patients followed *qd* and *bid* dosing, respectively, at Week 24.

### SADRs, SAEs, and hypoglycemia

There were no SADRs, SAEs, or major hypoglycemic events reported in either group throughout the study.

In the GLA group, 11.8% of patients reported overall hypoglycemia at baseline compared to 7.5% at Week 24 [Table 3]. The corresponding incidence rates of overall hypoglycemia were 2.80 events per patient-year and

**Table 1: Demographic and baseline characteristics by pre-study basal insulin regimen**

| Parameter | GLA group | NEU group |
|---|---|---|
| *n* | 102 | 39 |
| Sex, M/F (%) | 76.5/23.5 | 61.5/38.5 |
| Mean (SD) | | |
| Age, years | 54.3 (11.0) | 55.8 (8.4) |
| Body weight, kg | 75.6 (15.2) | 70.2 (13.0) |
| BMI, kg/m$^2$ | 28.3 (5.2) | 27.5 (4.4) |
| Diabetes duration, years | 9.0 (5.7) | 8.9 (6.3) |
| Insulin duration, years | 2.6 (2.0) | 2.5 (2.9) |
| $HbA_{1c}$, % | 9.9 (1.8) | 9.1 (1.2) |
| OGLDs at baseline, *n* (%) | | |
| Metformin | 73 (83.0) | 28 (77.8) |
| Sulfonylureas | 62 (70.5) | 24 (66.7) |
| Thiazolidinediones | 19 (21.6) | 6 (16.7) |
| One | 24 (27.3) | 14 (38.9) |
| Two | 47 (53.4) | 15 (41.7) |
| >Two | 17 (19.3) | 7 (19.4) |

BMI: Body mass index; F: Female; GLA: Pre-study insulin glargine to insulin detemir; $HbA_{1c}$: Glycated hemoglobin $A_{1c}$; M: Male; NEU: Pre-study neutral protamine Hagedorn insulin to insulin detemir; OGLD: Oral glucose-lowering drug

**Table 2: Insulin dose and frequency by pre-study basal insulin regimen**

| | GLA group | NEU group |
|---|---|---|
| Insulin dose, U/kg | | |
| *n* | 94 | 35 |
| Pre-study*† | 0.26 (0.13) | 0.27 (0.16) |
| Baseline* | 0.23 (0.09) | 0.24 (0.09) |
| Week 24* | 0.25 (0.12) | 0.28 (0.09) |
| Daily dose frequency | | |
| Pre-study | | |
| *n* | 102 | 39 |
| Once# | 86 (84.3) | 25 (64.1) |
| Twice# | 16 (15.7) | 14 (35.9) |
| Baseline | | |
| *n* | 102 | 39 |
| Once# | 89 (87.3) | 34 (87.2) |
| Twice# | 12 (11.8) | 5 (12.8) |
| Thrice# | 1 (1.0) | 0 |
| Week 24 | | |
| *n* | 67 | 31 |
| Once# | 42 (62.7) | 21 (67.7) |
| Twice# | 25 (37.3) | 10 (32.3) |

GLA: Pre-study insulin glargine to insulin detemir; NEU: Pre-study neutral protamine Hagedorn insulin to insulin detemir, *Data are mean (SD), †Dose in IU/kg for neutral protamine Hagedorn insulin at pre-study, #Data are *n* (%)

1.36 events per patient-year, respectively. No nocturnal hypoglycemic events were reported at Week 24 in the GLA group [Table 3].

At baseline in the NEU group, 10.3% of patients reported overall hypoglycemia, corresponding to an incidence rate of 2.33 events per patient-year [Table 3]. At Week 24, no hypoglycemic event of any category was reported in the NEU group.

### Glycemic parameters

In the GLA group, the mean ± SD $HbA_{1c}$ was 9.9 ± 1.8% at baseline compared to 7.6 ± 0.9% at Week 24 (mean change: −2.3 ± 1.7%). In the NEU group, the mean $HbA_{1c}$ level was 9.1 ± 1.2% at baseline and 7.3 ± 0.7% at Week 24 (mean change: −1.7 ± 0.9%).

Fifteen patients had $HbA_{1c}$ levels of <7.0% at Week 24 in the GLA group compared to 3 patients at baseline. In the NEU group, 6 patients had $HbA_{1c}$ levels of <7.0% at Week 24 compared to 1 patient at baseline.

The mean FPG and PPPG levels also appeared to improve from baseline to Week 24 in the GLA and NEU groups [Table 4].

### Lipids, body weight, and SBP

The mean levels of total cholesterol, triglycerides, and LDL cholesterol appeared to improve over 24 weeks in the GLA and NEU groups, while the mean HDL cholesterol levels appeared unchanged in both groups [Table 5].

In the GLA group, the mean body weight was 72.1 ± 11.7 kg at baseline and 72.3 ± 11.3 kg at Week 24 [Table 5]. Over the same period in the NEU group, the mean body weight was 69.5 ± 13.0 kg and 69.8 ± 12.4 kg, respectively.

**Table 3: Outcomes for hypoglycemia by pre-study basal insulin regimen**

| Event per patient-year/percent with at least one event | GLA group | NEU group |
|---|---|---|
| Overall | | |
| Baseline | 2.80/11.8 | 2.33/10.3 |
| Week 24 | 1.36/7.5 | 0/0 |
| Nocturnal | | |
| Baseline | 1.91/10.8 | 0.33/2.6 |
| Week 24 | 0/0 | 0/0 |
| Minor | | |
| Baseline | 2.80/11.8 | 1.33/10.3 |
| Week 24 | 1.36/7.5 | 0/0 |
| Major | | |
| Baseline | 0/0 | 1.0/2.6 |
| Week 24 | 0/0 | 0/0 |

GLA: Pre-study insulin glargine to insulin detemir; NEU: Pre-study neutral protamine Hagedorn insulin to insulin detemir

At baseline and Week 24, the mean SBP was 131.6 ± 13.8 mmHg and 128.5 ± 15.7 mmHg, respectively, in the GLA group, and 140.5 ± 16.0 mmHg

**Table 4: Glycemic parameters by pre-study basal insulin**

| | GLA group | NEU group |
|---|---|---|
| $HbA_{1c}$, % | | |
| *N* | 61 | 30 |
| Baseline | 9.9 (1.8) | 9.1 (1.2) |
| Week 24 | 7.6 (0.9) | 7.3 (0.7) |
| Change | −2.3 (1.7) | −1.7 (0.9) |
| FPG, mg/dL | | |
| *n* | 56 | 29 |
| Baseline | 193.3 (47.5) | 168.1 (59.5) |
| Week 24 | 138.5 (39.4) | 130.0 (37.3) |
| Change | −54.7 (38.6) | −38.0 (47.6) |
| PPPG, mg/dL | | |
| *n* | 41 | 18 |
| Baseline | 298.6 (68.7) | 263.2 (68.4) |
| Week 24 | 239.6 (65.1) | 236.2 (70.9) |
| Change | −59.0 (60.9) | −27.0 (38.5) |

FPG: Fasting plasma glucose; GLA: Pre-study insulin glargine to insulin detemir; $HbA_{1c}$: Glycated hemoglobin $A_{1c}$; NEU: Pre-study neutral protamine Hagedorn insulin to insulin detemir; PPPG: Postprandial plasma glucose, Baseline, Week 24 and change values are mean (SD)

**Table 5: Outcomes for lipid profile, body weight and SBP by pre-study basal insulin regimen**

| | GLA group | NEU group |
|---|---|---|
| Total cholesterol, mmol/L | | |
| *N* | 11 | 5 |
| Baseline | 5.5 (1.1) | 4.9 (1.1) |
| Week 24 | 5.1 (1.0) | 4.6 (0.9) |
| Change | −0.3 (0.3) | −0.3 (0.2) |
| Triglycerides, mmol/L | | |
| *N* | 14 | 7 |
| Baseline | 2.0 (0.8) | 1.7 (0.6) |
| Week 24 | 1.9 (0.8) | 1.5 (0.6) |
| Change | −0.1 (0.2) | −0.3 (0.5) |
| HDL cholesterol, mmol/L | | |
| *N* | 15 | 9 |
| Baseline | 1.0 (0.2) | 1.1 (0.2) |
| Week 24 | 0.9 (0.2) | 1.1 (0.1) |
| Change | −0.0 (0.2) | −0.0 (0.3) |
| LDL cholesterol, mmol/L | | |
| *N* | 14 | 9 |
| Baseline | 2.9 (1.0) | 3.2 (1.0) |
| Week 24 | 2.7 (0.8) | 2.8 (0.9) |
| Change | −0.2 (0.3) | −0.4 (0.5) |
| Body weight, kg | | |
| *N* | 60 | 26 |
| Baseline | 72.1 (11.7) | 69.5 (13.0) |
| Week 24 | 72.3 (11.3) | 69.8 (12.4) |
| Change | 0.2 (6.3) | 0.3 (4.0) |
| SBP, mmHg | | |
| *N* | 39 | 18 |
| Baseline | 131.6 (13.8) | 140.5 (16.0) |
| Week 24 | 128.5 (15.7) | 132.8 (12.2) |
| Change | −3.1 (14.9) | −7.7 (16.7) |

GLA: Pre-study insulin glargine to insulin detemir; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; NEU: Pre-study neutral protamine Hagedorn insulin to insulin detemir; SBP: Systolic blood pressure, Baseline, Week 24 and change values are mean (SD). *P* values are not presented as the number of patients analyzed was less than 100

and 132.8 ± 12.2 mmHg, respectively, in the NEU group [Table 5].

### *QoL*

In the GLA group, the mean QoL score on the EQ-VAS was 56.2 ± 10.9 points at baseline compared to 72.4 ± 10.5 points at Week 24. In the NEU group, the mean QoL score was 56.6 ± 6.7 points at baseline and 73.8 ± 9.9 points at Week 24.

## Discussion

This sub-analysis demonstrated the clinical safety and effectiveness of IDet therapy in Indian patients with T2D switched from IGlar and NPH insulin. As observed in the overall A$_1$chieve study,[14] IDet therapy appeared to be associated with beneficial effects on glycemic parameters and hypoglycemia.

Glycemic control was sub-optimal at baseline in patients previously treated with IGlar and NPH insulin. In the GLA group, patients had a mean baseline HbA$_{1c}$ of 9.9 ± 1.8%, diabetes duration of 9.0 ± 5.7 years and had been taking insulin for an average of 2.6 ± 2.0 years. In the NEU group, the mean baseline HbA$_{1c}$ level was 9.1 ± 1.2%, diabetes duration was 8.9 ± 6.3 years and patients had been on insulin therapy for an average of 2.5 ± 2.9 years.

Switching to IDet therapy was seen to be well-tolerated in the GLA and NEU groups with no SADRs, SAEs, or major hypoglycemic events reported during the study. Clinical trials have not identified any difference between IDet and IGlar with regard to the risk of hypoglycemia.[7,8] In this sub-analysis, the incidence rate of overall hypoglycemia was 2.80 events per patient-year at baseline and 1.36 events per patient-year at Week 24 in the GLA group. No nocturnal hypoglycemic events were reported at Week 24 in the GLA group against an incidence rate of 1.91 events per patient-year at baseline.

IDet has been noted to have a significantly lower risk association for hypoglycemia than NPH insulin in 2 randomized controlled trials in patients with T2D.[9,15] In the NEU group in this study, no events of hypoglycemia were reported at Week 24 against a baseline incidence rate of 2.33 events per patient-year.

Glycemic parameters appeared to improve after 24 weeks in both the GLA and NEU groups. At Week 24, the mean HbA$_{1c}$ was 7.6 ± 0.9% in the GLA group and 7.3 ± 0.7% in the NEU group. However, these levels still fall short of the internationally recommended HbA$_{1c}$ target of <7.0%.[16] Also, the number of patients meeting the HbA$_{1c}$ target of <7.0% at Week 24 was low in both groups.

Most patients (>60%) in both the GLA and NEU groups followed *qd* dosing of IDet at Week 24, with the remaining patients following *bid* dosing. In the GLA group, the mean daily insulin dose by weight was 0.23 ± 0.09 U/kg at baseline and 0.25 ± 0.12 U/kg at Week 24. In the NEU group also, the difference in mean dose between baseline and Week 24 was small (0.24 ± 0.09 and 0.28 ± 0.09, respectively), suggesting that perhaps the physicians were overly cautious in applying dose titration. It is possible that a more aggressive approach to dose titration may have led to more effective reductions in glycemic levels and more patients achieving the recommended HbA$_{1c}$ target at Week 24. A general lack of insulin dose optimization in clinical practice in India has also been identified in the prospective 26-week IMPACT study, which evaluated the effectiveness of the Indian insulin guideline on premixed insulin.[17]

Mean body weights remained very similar from baseline to Week 24 in both the GLA and NEU groups, in line with the known weight-sparing effect of IDet therapy.[7,15] The mean QoL also appeared to improve with IDet therapy in both groups, in line with the overall A$_1$chieve study results[14] that showed a significant improvement in QoL in patients treated with IDet.

This study may have been limited by the lack of a control group and the possible introduction of recall bias in the reporting of safety data, particularly hypoglycemic events. The small number of patients in this sub-analysis was also a limiting factor. Concomitant medication and diet were not restricted; hence, it was not possible to determine the effect, if any, of non-pharmacological treatment on the study outcomes. The heterogeneity of the local healthcare systems meant that there was no standardization across sites. However, this study provided the opportunity to study the effect of switching therapy from NPH insulin and IGlar to IDet in the routine clinical setting in India. All measurements made by the laboratories followed local standardization procedures and the 24-week duration of the study was considered adequate to determine the preliminary clinical trend following therapy change.

Data from non-interventional studies such as A$_1$chieve can help bridge the gap between recommended healthcare policies for T2D and actual clinical practice. Also, patient education can play a key role in improving compliance to blood glucose monitoring, weight control, diet and exercise. A holistic approach to the treatment of T2D, involving increased patient education, recommendations for diet and exercise and therapeutic strategies that target glycemic levels without increasing the risk of hypoglycemia and weight gain, may be indicated to improve patient compliance and facilitate disease management.[18] In this sub-analysis,

IDet therapy was noted to be well-tolerated with minimal change in weight and a low risk of hypoglycemia in patients switched from NPH insulin and IGlar. It would be interesting to note the effect of additional dose titration with IDet over a further period of evaluation in a larger patient group.

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