Real-World Efficacy and Safety of Dulaglutide in Korean Patients with Type 2 Diabetes Mellitus: A Retrospective Study in a Tertiary Referral Center

Jee Hee Yoon, A Ram Hong*, Wonsuk Choi, Ji Yong Park, Hee Kyung Kim, and Ho-Cheol Kang
Department of Internal Medicine, Chonnam National University Medical School, Gwangju, Korea

This study was conducted to evaluate the efficacy and safety of once-weekly dulaglutide therapy as add-on to oral antidiabetic drugs (OADs) and basal insulin in Korean patients with type 2 diabetes mellitus (T2DM) in real-world clinical practice. We retrospectively reviewed the medical records of 112 patients who received dulaglutide in a tertiary referral center. The primary efficacy endpoint was a change in glycated hemoglobin (HbA1c) between baseline and 6 months. The secondary endpoints were the percentage of patients achieving HbA1c <7.0% or ≤6.5% and the change of body weight at 6 months. At baseline, the mean HbA1c was 8.7 % (8.8% in the OAD combination and 8.5% in the basal insulin combination group). The mean adjusted HbA1c at 6 months decreased by −1.13% in all patients (p<0.001), and by −1.36 and −0.74% in the OAD combination and basal insulin combination group, respectively. A significant reduction of −2.9 kg in body weight was observed in all patients at 6 months (p<0.001). Approximately 34.8% and 23.2% of patients achieved HbA1c <7.0% and ≤6.5%, respectively. Higher baseline HbA1c and no previous insulin therapy were associated with positive responses to dulaglutide on multivariate analysis. Mild gastrointestinal issues (23.2%) were the most frequently observed adverse events. Dulaglutide is an effective and durable treatment option as OAD and basal insulin combination therapy in Korean patients with T2DM.

Key Words: Dulaglutide; Diabetes Mellitus, Type 2; Blood Glucose

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is emerging as a prevalent disease worldwide and affects 14.4% of adults aged 30 or older in Korea.1 A wide range of glucose-lowering agents has been developed to achieve glycemic target goals in T2DM, which is a chronic, progressive, metabolic disorder. The American Diabetes Association and the European Association for the Study of Diabetes recommend metformin as the first-line therapy for T2DM.2,3 When additional antidiabetic therapies are required to achieve the glycemic target, an individualized, patient-based approach, with due consideration to the presence of atherosclerotic cardiovascular disease and chronic kidney disease, body weight, hypoglycemia, and cost-effectiveness, is recommended. The glucagon-like peptide-1 (GLP-1) receptor agonist (RA) is preferred as the second-line therapeutic agent in patients who need adjutant antidiabetic therapy. Moreover, GLP-1 RAs are preferred injectable therapies for insulin in patients with inadequate glycemic control with oral antidiabetic drugs (OADs).

GLP-1 is an incretin hormone, which increases glucose load-based insulin secretion and decreases glucagon secretion, and thereby slows gastric emptying and reduces appetite.4 The GLP-1 RAs have demonstrated glucose-lowering efficacy with a low risk of hypoglycemia as well as marked body weight reduction in several randomized controlled trials (RCTs).5,6 In addition, treatment with GLP-1 RA has showed a significant reduction of major cardiovascular disease risk and mortality, and improved renal outcomes.8

Dulaglutide is a long-acting human GLP-1 RA that has
been approved for the treatment of T2DM, and is administered weekly. Since its introduction, the usage of dulaglutide has continuously increased in Korea. The initiation of dulaglutide therapy can be considered in various clinical settings; however, given concerns associated with insurance coverage, dulaglutide is generally used in combination therapy with OAD or basal insulin. Several recent real-world studies have shown consistent glucose-lowering efficacy with body weight reduction in patients with T2DM treated with dulaglutide when compared with the results from previous RCTs.9,10 Similar findings from Korean patients with T2DM were reported in two studies that evaluated the glycemic efficacy of dulaglutide for 6 months.11,12 However, little is known about the differences in glucose-lowering efficacy of dulaglutide according to the background glucose-lowering regimen.

Therefore, this study aimed to evaluate the efficacy and safety of dulaglutide as OAD and basal insulin combinations in a real-world clinical setting in Korean patients with T2DM.

MATERIALS AND METHODS

1. Study population

We retrospectively reviewed the medical records of patients with T2DM who started dulaglutide therapy at Chonnam National University Hwasun Hospital between January 2017 and August 2019. This study included patients with T2DM who received dulaglutide for more than 6 months. We excluded patients who had been previously treated with other types of GLP-1 RA, those who had self-reported low adherence, and received high-dose steroid therapy and chemotherapy during dulaglutide therapy for the analysis. We further excluded the patients who experienced significant changes in treatment regimens, such as the addition of insulin or change in OAD class at the initiation of dulaglutide or during the treatment. A total of 112 patients were included in the final analysis. The patients were divided into two groups according to the background glucose-lowering therapy: the OAD combination group (i.e., dulaglutide in combination with OADs) and the basal insulin combination group (i.e., dulaglutide in combination with basal insulin). The OAD combination group was further subdivided into two groups based on the history of insulin therapy prior to the initiation of dulaglutide. Meanwhile, the basal insulin combination group was further subdivided into two groups by the history of bolus insulin therapy prior to the initiation of dulaglutide therapy.

This study was conducted in accordance with the Declaration of Helsinki and was reviewed by the Institutional Review board of Chonnam National University Hwasun Hospital (IRB No.: CNUHH-2019-085). The need for informed consent was waived due to the retrospective nature of the study.

2. Efficacy and safety assessment

For the evaluation of clinical characteristics, we collected data on age, sex, body weight, body mass index (BMI), duration of diabetes, total daily insulin dose, and comorbidities including hypertension, dyslipidemia, and cardiovascular diseases. The HbA1c measurements were conducted at baseline, and repeated after 3 months (±4 weeks) and 6 months (±4 weeks) following dulaglutide initiation. We further collected data on the levels of fasting plasma glucose (FPG), total cholesterol, triglyceride, high-density lipoprotein-cholesterol, low-density lipoprotein-cholesterol, liver enzymes, serum creatinine, and estimated glomerular filtration rate (eGFR).

The primary efficacy endpoint was a change in HbA1c between baseline and 6 months. The secondary efficacy endpoints were the percentage of patients who achieved a glycemic target of HbA1c <7.0% or ≤6.5% at 6 months as well as the change in body weight between baseline and 6 months. Additionally, we investigated the clinical characteristics of patients who achieved a good response to dulaglutide, which was defined as a decrease in HbA1c of more than 1.0% after 6 months of dulaglutide therapy.12 We further examined a response to dulaglutide according to the duration of diabetes, BMI, and eGFR.

The self-reported adverse events including hypoglycemia, gastrointestinal problems, and injection-related reactions were collected from a review of the medical records.

3. Statistical analysis

Continuous variables were expressed as the mean±standard deviation and categorical variables were expressed as the number (%). The chi-square test was used for the comparison of categorical variables. The paired t-test was used to compare the changes in HbA1c and body weight between baseline and 6 months in each treatment group. Intergroup comparisons were undertaken with the analysis of covariance after adjusting for the baseline levels. The changes in each parameter were expressed as the adjusted least square (LS) means and standard error. Univariate and multivariate logistic regression analyses were conducted to evaluate the predictive factors for the therapeutic response to dulaglutide. Risk was reported as odds ratios and 95% confidence intervals (CIs). All statistical analyses were performed using SPSS software version 25.0 for Windows (IBM Corp., Armonk, NY, USA). p-values <0.05 were considered to indicate statistical significance.

RESULTS

1. Baseline characteristics of the study population

The baseline characteristics of the study participants are presented in Table 1. The mean age was 59.7 years and 35.7% of them were female. The mean body weight was 77.9 kg and BMI was 27.7 kg/m². The mean duration of diabetes was 12.2 years in the entire study cohort and was significantly longer in the basal insulin combination group than in the OAD combination group (p=0.015). Before the
TABLE 1. Baseline characteristics of study population

|                          | Overall (n=112) | OAD combination (n=72) | Basal insulin combination (n=40) | p value |
|--------------------------|-----------------|-----------------------|----------------------------------|---------|
| **Age, yr**              | 59.7±11.6       | 58.7±11.9             | 61.3±10.9                        | 0.259   |
| **Female sex**           | 40 (35.7)       | 26 (36.1)             | 14 (35.0)                        | 0.906   |
| **Height, cm**           | 166.5±8.1       | 166.9±8.3             | 165.8±7.8                       | 0.513   |
| **Body weight, kg**      | 77.9±16.0       | 79.3±17.5             | 75.3±12.6                       | 0.225   |
| **Body mass index, kg/m²** | 27.7±4.2       | 27.9±4.3              | 27.2±4.1                        | 0.416   |
| **Duration of diabetes, yr** | 12.2±8.6     | 10.7±7.9              | 14.9±9.3                        | 0.015   |
| **Systolic blood pressure, mmHg** | 129.2±15.0     | 129.9±15.7            | 128.1±13.7                      | 0.552   |
| **Diastolic blood pressure, mmHg** | 74.9±10.8    | 76.6±10.6             | 71.8±10.7                       | 0.024   |
| **HbA1c, %**             | 8.7±1.4         | 8.8±1.5               | 8.5±1.4                         | 0.376   |
| **Fasting plasma glucose, mg/dL** | 180.1±67.0   | 190.1±70.6            | 159.9±54.8                      | 0.037   |
| **Total cholesterol, mg/dL** | 155.7±43.3   | 160.0±37.0            | 147.7±52.7                      | 0.175   |
| **Triglyceride, mg/dL**  | 154.7±92.8      | 150.1±87.0            | 163.8±103.9                     | 0.486   |
| **HDL cholesterol, mg/dL** | 43.0±9.1       | 42.9±8.7              | 43.4±10.0                       | 0.807   |
| **LDL cholesterol, mg/dL** | 86.6±30.4      | 89.9±27.1             | 80.1±35.4                       | 0.128   |
| **Aspartate aminotransferase, U/L** | 30.2±18.6     | 32.9±21.5             | 24.9±9.5                        | 0.034   |
| **Alanine aminotransferase, U/L** | 34.2±26.0    | 35.8±30.3             | 25.8±10.9                       | 0.012   |
| **Serum creatinine, mg/dL** | 1.05±0.91      | 1.05±0.76             | 1.06±0.42                       | 0.981   |
| **Estimated glomerular filtration rate, mL/min/1.73 m²** | 81.5±30.3      | 83.8±30.0             | 77.2±31.0                       | 0.400   |
| **Hypertension**         | 65 (58.0)       | 38 (52.8)             | 71 (77.5)                       | 0.130   |
| **Dyslipidemia**         | 82 (73.2)       | 51 (70.8)             | 31 (77.5)                       | 0.445   |
| **Cardiovascular disease** | 22 (19.6)      | 15 (20.8)             | 7 (17.5)                        | 0.671   |
| **Dulaglutide dose**     |                |                      |                                  |        |
| 0.75 mg                  | 29 (25.9)       | 11 (15.3)             | 18 (45.0)                       |        |
| 1.5 mg                   | 83 (74.1)       | 61 (84.7)             | 22 (55.0)                       |        |
| **Treatment before dulaglutide initiation** |                |                      |                                  |        |
| **Insulin therapy**      | 52 (46.4)       | 12 (16.7)             | 40 (100.0)                      | <0.001  |
| **Daily insulin dose, IU/day** | 44.7±29.7     | 20.9±10.1             | 51.8±30.0                       | 0.001   |
| **OAD**                  |                |                      |                                  |        |
| **Metformin**            | 104 (92.9)      | 67 (93.1)             | 37 (92.5)                       | 0.913   |
| **Sulfonylurea**         | 66 (58.9)       | 47 (65.3)             | 19 (47.5)                       | 0.067   |
| **DPP4 inhibitor**       | 73 (65.2)       | 49 (68.1)             | 24 (60.0)                       | 0.391   |
| **Thiazolidinedione**    | 13 (11.6)       | 10 (13.9)             | 3 (7.5)                         | 0.312   |
| **SGLT2 inhibitor**      | 9 (8.0)         | 7 (9.7)               | 2 (5.0)                         | 0.378   |
| **Alpha-glucosidase inhibitor** | 2 (1.8)       | 1 (1.4)               | 1 (2.5)                         | 0.671   |

Values are expressed as mean±standard deviation or number (%).
Hypertension is defined as a systolic blood pressure ≥ 140 mmHg, or diastolic blood pressure ≥ 90 mmHg, or taking anti-hypertensive medication. Dyslipidemia is defined as taking lipid-lowering agents including statin, ezetimibe, fibrate, and omega-3.
OAD: oral anti-diabetic drug, HDL: high-density lipoprotein, LDL: low-density lipoprotein, DPP4: Dipeptidyl peptidase-4, SGLT2: sodium-glucose cotransporter-2.

initiation of dulaglutide therapy, the majority of patients had received metformin. Approximately 46.4% of the patients were treated with insulin before the initiation of dulaglutide therapy, and the mean total daily insulin dose was 44.7 IU. At the initiation of dulaglutide therapy, approximately 29.9 IU of insulin switched to dulaglutide in the OAD combination group. The mean total daily insulin dose was 51.8 IU in the basal insulin combination group. Six patients titrated their insulin doses (>10% of the baseline) at the initiation of dulaglutide.

At baseline, the mean HbA1c was 8.7% in all patients, 8.8% in the OAD combination group and 8.5% in the basal insulin combination group. The mean FPG concentration was higher in the OAD combination group than that in the basal insulin combination group (190.1 mg/dL vs. 159.9 mg/dL, p=0.037). There were no significant differences in the baseline fasting lipid profile, serum creatinine, and estimated glomerular filtration rate (GFR) levels between the two groups. Although the levels of aspartate aminotransferase and alanine aminotransferase were higher in the OAD combination group than in the basal insulin combination group (p=0.034 and p=0.012, respectively), both parameters were within the reference ranges. The prevalence of hypertension, cardiovascular disease, and dyslipidemia did not differ between the two groups. Approximately 74.1% of all patients received 1.5 mg dulaglutide; the OAD combination group was associated with higher dose of dulaglutide than the basal insulin combination group (p=0.001).
2. Efficacy

At 6 months, dulaglutide therapy exhibited a significant reduction in HbA1c levels of $-1.13\%$ (95% CI, $-1.38$ to $-0.89$; $p<0.001$) in all patients. Nineteen patients (17.0\%) showed worsening of HbA1c at 6 months after dulaglutide therapy compared with the baseline HbA1c. The mean adjusted HbA1c decreased by $-1.36\%$ (95% CI, $-1.61$ to $-1.10$) in the OAD combination group and $-0.74\%$ (95% CI, $-1.08$ to $-0.40$) in the basal insulin combination group ($p<0.001$ and $p=0.001$, respectively). The difference in the LS mean change between the two groups was $-0.62\%$ (95% CI, $-1.04$ to $-0.19$; $p=0.006$; Fig. 1A). There was a significant reduction of $-2.9$ kg in the body weight in all patients ($p<0.001$), and of $-3.5$ kg in the OAD combination group ($p<0.001$); however, a statistically significant difference was not observed in the basal insulin combination group ($-1.8$ kg, $p=0.068$) (Fig. 1B).

The proportion of patients who achieved HbA1c <7.0\% at 6 months was 34.8\% in all patients, 41.7\% in the OAD combination group, and 22.5\% in the basal insulin combination group ($p=0.041$). Moreover, with the HbA1c ≥6.5\% as the glycemic target, 23.2\% of all patients achieved the glycemic target goal (31.9\% in the OAD combination group and 7.5\% in the basal insulin combination group, $p=0.003$; Fig. 1C).

In the OAD combination group, there was no significant difference in the glucose-lowering efficacy between the insulin-naïve and previous insulin therapy group (LS mean change $-1.39\%$ and $-1.47\%$, respectively; between-treatment difference in HbA1c of 0.080 [95% CI, $-0.620$ to $0.779$]; $p=0.821$; Table 2). Similarly, in the basal insulin combination group, the magnitude of the HbA1c reduction did not differ between the bolus insulin-naïve and previous bolus insulin therapy group (LS mean change $-0.80$ and $-0.68\%$, respectively; between-treatment difference of $-0.217$ [95% CI, $-0.930$ to $0.497$]; $p=0.542$). The total daily insulin dose decreased by $-20.7$ IU in the basal insulin combination group ($p<0.001$), and by $-16.0$ and $-23.5$ in the bolus insulin-naïve and previous bolus insulin therapy group, respectively.

Overall, 60 patients (53.6\%) were categorized as good responders (HbA1c reduction ≥1.0\%) after 6 months of dulaglutide therapy. Shorter duration of diabetes and higher baseline glycemic parameters (HbA1c and FPG) were associated with a good response to dulaglutide. However, patients who had a history of insulin therapy showed a poor response to dulaglutide (Table 3). Logistic regression analyses were used for the identification of predictable parameters for the glucose-lowering effect of dulaglutide. A univariate regression analysis showed that shorter duration

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**FIG. 1.** Changes in (A) HbA1c and (B) body weight between baseline and 6 months of treatment with dulaglutide. (C) Proportion of patients who achieved glycemic target goals of HbA1c <7.0\% or ≤6.5\%. Data are expressed as mean (standard deviation) or %. OAD, oral anti-diabetic drug. *p<0.05 between OAD combination and basal insulin combination group.
TABLE 2. Changes in HbA1c after 6 months of treatment with dulaglutide as OAD combination and basal insulin combination

| Variable                        | Baseline | 6 months | LS mean change from baseline (95% CI) | Difference in LS mean change (95% CI) |
|---------------------------------|----------|----------|--------------------------------------|--------------------------------------|
| OAD combination                 |          |          |                                      |                                      |
| Overall (n=72)                  | 8.8±0.1  | 7.4±0.1  | -1.36 (-1.61 to -1.10)a               |                                      |
| Insulin-naïve (n=60)            | 8.8±0.2  | 7.4±0.2  | -1.39 (-1.67 to -1.10)a               | 0.080 (-0.620 to 0.779)             |
| Previous insulin therapy (n=12) | 9.0±0.5  | 7.4±0.4  | -1.47 (-2.10 to -0.83)a               |                                      |
| Basal insulin combination       |          |          |                                      |                                      |
| Overall (n=40)                  | 8.5±0.2  | 7.9±0.2  | -0.74 (-1.08 to -0.40)a               |                                      |
| Bolus insulin-naïve (n=15)      | 8.7±0.3  | 7.9±0.4  | -0.80 (-1.36 to -0.23)a               | -0.217 (-0.930 to 0.497)            |
| Previous bolus insulin therapy  | 8.4±0.3  | 7.9±0.3  | -0.68 (-1.01 to -0.14)a               |                                      |

Values are presented as mean±standard error. LS: least square, CI: confidence interval. *p<0.05 from baseline in overall patients and each treatment subgroup.

TABLE 3. Clinical characteristics between the good responder and the poor responders in all patients

| Variable                        | Good responders (n=60) | Poor responders (n=52) | p value |
|---------------------------------|-----------------------|-----------------------|---------|
| Age, yr                         | 58.9±11.4             | 60.5±11.9             | 0.478   |
| Female sex                      | 22 (36.7)             | 18 (34.6)             | 0.821   |
| Body weight, kg                 | 78.6±17.5             | 77.0±14.2             | 0.590   |
| Body mass index, kg/m²          | 27.8±4.3              | 27.5±4.2              | 0.768   |
| Duration of diabetes, yr        | 10.2±8.0              | 14.6±8.7              | 0.008   |
| Hypertension                    | 39 (65.0)             | 26 (50.0)             | 0.109   |
| Dyslipidemia                    | 40 (66.7)             | 42 (80.8)             | 0.093   |
| Cardiovascular disease          | 12 (20.0)             | 10 (19.2)             | 0.919   |
| Diabetic retinopathy            | 16 (26.7)             | 15 (28.8)             | 0.327   |
| Baseline HbA1c, %               | 9.3±1.3               | 8.0±1.3               | <0.001  |
| Baseline fasting plasma glucose, mg/dL | 194.4±65.2          | 163.1±65.7            | 0.022   |
| Previous insulin therapy before dulaglutide initiation | 20 (33.3)             | 32 (61.5)             | 0.003   |

Values are presented as mean±standard deviation or number (%).

of diabetes, higher baseline HbA1c, and no previous insulin use were associated with a good response to dulaglutide at 6 months. On multivariate analysis, higher baseline HbA1c and no previous insulin therapy remained statistically significant (Table 4).

We further analyzed the reduction of HbA1c in all patients according to the duration of diabetes <10 years or ≥10 years, BMI <25 kg/m² or ≥25 kg/m², and eGFR <60 mL/min/1.73 m² or ≥60 mL/min/1.73 m². There were no significant differences in the reduction of HbA1c with respect to the duration of diabetes, BMI, and eGFR (p=0.177, p=0.158, p=0.606, respectively) (Fig. 2).

3. Safety profiles

Adverse events were reported in 44 patients (39.3%) during the 6 months of dulaglutide therapy. The most common adverse events were gastrointestinal problems, including nausea, vomiting, dyspepsia, and abdominal pain (23.2%). Hypoglycemia was observed in 15 patients (13.4%), which was associated with the use of sulfonylurea (n=7) and basal insulin (n=8) as background therapy. Severe hypoglycemia was not reported in the study population during the dulaglutide therapy. Four patients (3.6%) had injection-site reactions that were self-limited.

TABLE 4. Logistic regression analysis to predict good responders to dulaglutide in all patients

| Variable                        | Odd ratio | 95% CI     | p value |
|---------------------------------|-----------|------------|---------|
| Univariate analysis             |           |            |         |
| Age, yr                         | 0.99      | 0.96-1.02  | 0.474   |
| Female sex                      | 0.91      | 0.42-1.99  | 0.821   |
| Body weight, kg                 | 1.01      | 0.98-1.03  | 0.594   |
| Body mass index, kg/m²          | 1.01      | 0.93-1.11  | 0.766   |
| Duration of diabetes, yr        | 0.94      | 0.90-0.99  | 0.010   |
| Baseline HbA1c, %               | 2.25      | 1.58-3.20  | <0.001  |
| No previous insulin therapy     | 3.20      | 1.48-6.94  | 0.003   |
| Hypertension                    | 0.54      | 0.25-1.15  | 0.110   |
| Cardiovascular disease          | 0.95      | 0.37-2.43  | 0.919   |
| Multi-variate analysis          |           |            |         |
| Duration of diabetes, yr        | 0.96      | 0.90-1.01  | 0.108   |
| HbA1c, %                        | 2.44      | 1.61-3.70  | <0.001  |
| No previous insulin therapy     | 4.27      | 1.62-11.3  | 0.003   |

CI: confidence interval.

DISCUSSION

In this analysis of real-world clinical data, we demonstrated that dulaglutide significantly improved the glyco-
mic control in Korean patients with T2DM. The glucose-lowering efficacy of dulaglutide was observed both in adding to OAD or adding to basal insulin over a 6-month period. Moreover, dulaglutide induced significant reduction in body weight. Our findings are similar to those of previous RCTs and real-world clinical studies in patients with T2DM who received dulaglutide treatment.

In the present study, dulaglutide exhibited a significant reduction in HbA1c of −1.13% at 6 months, which was consistent with the results from clinical trials and similar or slightly greater than that from real-world clinical studies conducted in Western countries. This may be associated with the pharmacokinetic and pharmacodynamic differences of GLP-1 RA in Caucasian and Asians, wherein Asian patients have higher postprandial glucose and postprandial excursion related to impaired β-cell function. We further analyzed the efficacy of dulaglutide separately as OAD combination or basal insulin because these two regimens are generally accepted in clinical practice. The glucose-lowering efficacy of dulaglutide therapy was observed in both groups. However, the magnitude of HbA1c reduction was greater in the OAD combination than in the basal insulin combination group (−1.36% vs −0.74%). Comparable glycemic efficacy of dulaglutide was reported in previous RCTs with dulaglutide in combination with OADs. The HbA1c-lowering effect of dulaglutide 1.5 mg was −1.42% at 26 weeks with metformin in the Assessment of Weekly AdministrRation of LY2189265 in Diabetes-6 (AWARD-6) trial and −1.40% with sulfonylurea in the AWARD-8 trial.

The observed glucose-lowering efficacy of dulaglutide as a basal insulin combination (−0.74%) was lower than those previously reported in the AWARD-9 trial. In the AWARD-9 trial, the mean HbA1c changes from baseline was −1.44% at 28 weeks in T2DM patients after being add-
Similar to previous studies, all reported cases of hypoglycemia were mild, and no severe hypoglycemia was documented in this study. In this study’s cohort, 3.6% of patients had an injection-site reaction; however, the incidence gradually decreased with repeated injections. Our data supported the safety profiles of dulaglutide in a clinical management plans that were comprised of various glucose-lowering regimens.

There are several limitations in the present study due to its retrospective design. The total cohort sample was small, in particular, for the analysis of glucose-lowering efficacy with regard to the background treatment regimens. Moreover, several parameters, including FPG, lipid profiles, blood pressure, and insulin sensitivity or resistance, were unavailable for an evaluation of the changes after dulaglutide therapy. Furthermore, safety profiles were not collected systematically; therefore, the self-report system might have resulted in under- and under-estimation of adverse events. Nevertheless, this study provided meaningful data on the real-world efficacy of dulaglutide in combination with various background glucose-lowering regimens.

Our findings prove the real-world efficacy and safety profiles of dulaglutide as OAD combination and basal insulin therapy in Korean patients with T2DM. A beneficial effect of body weight reduction was also observed. Dulaglutide therapy could be considered in real-world practice, either as OAD combination or basal insulin therapy in Korean T2DM patients with inadequate glycemic control and/or need for weight reduction.

**CONFLICT OF INTEREST STATEMENT**

None declared.

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