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The Effect of Liraglutide on Inflammatory Factors and Neurological Prognosis in Patients with Acute Cerebral Infarction Complicated with Type 2 Diabetes Mellitus

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Abstract: *Objective:* To investigate the effects of liraglutide on blood glucose reduction, inflammatory factor inhibition, vascular endothelial function improvement, and neurological function in patients with acute cerebral infarction (ACI) complicated with type 2 diabetes mellitus (T2DM). *Methods:* A total of 100 patients with ACI complicated with T2DM admitted to Tangshan People's Hospital from June 2022 to June 2025 were randomly divided into the liraglutide group and the control group, with 50 cases in each group. Both groups received routine treatment for the acute phase of cerebral infarction. On this basis, the control group was treated with insulin aspart and insulin glargine to control blood glucose, while the liraglutide group was treated with liraglutide combined with insulin glargine. Both groups were treated continuously for 3 months. *Results:* After treatment, the serum levels of tumor necrosis factor-α (TNF-α), interleukin-8 (IL-8), high-sensitivity C-reactive protein (hs-CRP), and interleukin-6 (IL-6) in the liraglutide group were significantly lower than those in the control group. The liraglutide group also reduced endothelin-1 (ET-1) levels and increased nitric oxide (NO) levels. Additionally, the National Institutes of Health Stroke Scale (NIHSS) score and modified Rankin Scale (mRS) score were significantly lower in the liraglutide group compared to the control group, with a higher total clinical effectiveness rate. *Conclusion:* Liraglutide improves neurological prognosis in patients with ACI complicated with T2DM by strengthening blood glucose reduction, alleviating inflammatory responses, and improving vascular endothelial function.

Keywords: Liraglutide; Cerebral infarction; Type 2 diabetes mellitus; Inflammatory factors; Neurological prognosis

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1. Introduction

Ischemic stroke is the most common type of stroke, accounting for 69.6% to 70.8% of all strokes in China. The treatment of ischemic stroke must adopt individualized regimens based on specific clinical symptoms, etiologies, and pathological characteristics, with classification being the core of individualized treatment. The commonly used TOAST classification is based on etiology and clinical manifestations. This study focused on the large artery atherosclerosis. T2DM is an independent risk factor for acute cerebral infarction. Patients with diabetic cerebral infarction often experience further

aggravation of neurological impairment due to stress-induced blood glucose fluctuations, and insulin resistance-mediated inflammatory responses and oxidative stress. Current research has confirmed that alleviating inflammatory response and improving vascular endothelial function are of significant importance for enhancing neurological prognosis. Liraglutide is a synthetic long-acting glucagon-like peptide-1 (GLP-1) analog. Recent animal studies have shown that liraglutide not only stabilizes blood glucose but also inhibits inflammatory responses, promotes cerebral angiogenesis and neurological function remodeling, and improves the prognosis of stroke patients. However, there are few reports on its clinical application effects. This study primarily aimed to investigate the effects of liraglutide on inflammatory factors and the improvement of neurological function in patients with complicated ACI with T2DM, so as to verify the efficacy of liraglutide, with the goal of providing evidence for its clinical application.

2. Objects and methods

2.1. Study objects

A total of 100 patients with first-onset acute cerebral infarction complicated with T2DM admitted to Tangshan People's Hospital from June 2022 to June 2025 were selected as the study subjects. Inclusion criteria: (1) Age > 18 years old; (2) Meet the diagnostic criteria for acute cerebral infarction in the "Chinese Guidelines for the Diagnosis and Treatment of Acute Ischemic Stroke" and confirmed as new cerebral infarction by cranial MRI + diffusion-weighted imaging; (3) Meet the diagnostic criteria for T2DM according to the "Chinese Guidelines for the Prevention and Treatment of Type 2 Diabetes Mellitus." (4) Onset time < 1 week; (5) NIHSS score of 5–15 points; (6) No anticoagulant, thrombolytic, or antifibrotic therapy before admission.

Exclusion criteria: (1) Complicated with cerebral hemorrhage or active bleeding. (2) Previous history of cerebral infarction; (3) Complicated with severe cardiopulmonary diseases, hepatic and renal insufficiency, or coagulation disorders. (4) Suffering from malignant tumors or mental illnesses. The enrolled patients were divided into the liraglutide group (experimental group) and the control group according to the random number table method, with 50 cases in each group. There were no significant differences in baseline clinical data between the two groups ($P > 0.05$), as shown in Table 1. This study was approved by the Ethics Committee of Tangshan People's Hospital (Approval No.: RMYY-LLKS-2025311). All patients and their families were informed of the study and signed the informed consent form.

2.2. Treatment methods

Neither group received intravenous thrombolysis or interventional therapy. Both groups received routine treatment for acute cerebral infarction, including antiplatelet aggregation, lipid-lowering and plaque stabilization. Based on the above treatment, the control group was treated with insulin aspart (subcutaneous injection before three meals) and insulin glargine (subcutaneous injection before bedtime) to control blood glucose. Patients in the liraglutide group received a combination therapy of bedtime subcutaneous injection of insulin glargine plus subcutaneous injection of liraglutide (Novo Nordisk A/S, Denmark; Registration No.: S20160004; Specification: 18 mg/3 mL). The initial dose of liraglutide was 0.6 mg once daily. If no adverse reactions occurred, the dose was increased to 1.2 mg/day after one week, followed by a gradual titration to 1.8 mg/day. In both groups, the dosage of insulin was adjusted based on blood glucose levels to maintain a fasting blood glucose (FBG) level below 7.0 mmol/L and a 2-hour postprandial blood glucose (2hPG) level below 11.1 mmol/L. The treatment duration for both groups was 3 months.

2.3. Observation indicators

2.3.1. Blood glucose and insulin resistance indicators

Peripheral venous blood samples were collected from patients in both groups before and after treatment to measure the levels of FBG, 2hPG, glycosylated hemoglobin (HbA1c), fasting insulin (FINS), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), total cholesterol (TC), and high-density lipoprotein cholesterol (HDL-C). The homeostasis

model assessment of insulin resistance (HOMA-IR) was calculated as HOMA-IR = FBG × FINS / 22.5.

2.3.2. Neurological function assessment

The NIHSS scale was used to assess the degree of neurological impairment, and the mRS was employed to evaluate neurological recovery in both groups before treatment and at 90 days after treatment.

2.3.3. Levels of inflammatory factor and vascular endothelial function

(1) Inflammatory Factor Levels: Serum levels of TNF-α, IL-8, and IL-6 were measured before treatment and at 3 months after treatment using a RaiseCare 12-plex cytokine detection kit (multiplex microsphere-based flow immunofluorescence assay) on a Mindray BriCyte E6 flow cytometer. And hs-CRP was measured using immunoturbidimetry.

(2) Vascular endothelial function: NO and ET-1 levels were measured before and after treatment using an enzyme-linked immunosorbent assay.

3. Statistical methods

Statistical analysis was carried out using SPSS software (Version 25.0). Continuous variables are reported as mean ± standard deviation (SD). For normally distributed continuous variables, comparisons between and within groups were made using the independent *t*-test and paired *t*-test, respectively. Non-normally distributed continuous variables are compared between groups using the Mann-Whitney U test. Categorical data are presented as n [%] and compared using the chi-square test or Fisher's exact test. Ordinal data are presented as n [%]; Intergroup comparisons are performed using the Mann-Whitney *U* test, while intragroup comparisons are performed using the Wilcoxon signed-rank test. A p-value < 0.05 is considered statistically significant.

Table 1. Comparison of general data between the control group and the Liraglutide group

	Control Group	Liraglutide Group	t/χ^2	p
Gender (n)			2.560	0.110
Male	21(42%)	29(58%)		
Female	29(58%)	21(42%)		
Hypertension (n)	9(18%)	6(12%)	0.706	0.401
Educational Background (n)			-0.835	0.403
Primary School	26(52%)	30(60%)		
Junior High School	21(42%)	18(36%)		
Senior High School and above	3(6%)	2(4%)		
Age (years old)	72.68 ± 7.64	72.34 ± 5.87	0.250	0.803
Smoking (n)	14(28%)	12(24%)	0.208	0.648
Drinking (n)	8(16%)	11(22%)	0.585	0.444

There were no significant differences in the levels of FDG, 2hPG, HbA1c, FINS, and HOMA-IR between the two groups before treatment ($P > 0.05$). After treatment, FDG, 2hPG, HbA1c, and HOMA-IR in both groups were lower than those before treatment, and the liraglutide group was significantly lower than the control group ($P < 0.05$, **Table 2**).

Table 2. Comparison of FDG, 2hPG, HbA1c, FINS, and HOMA-IR between the Two Groups Before and After Treatment

	FBG (mmol/L)		2hPG (mmol/L)		HbA1c (%)		FINS (uU/mL)		HOMA_IR	
	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment
Control Group	9.31 ± 1.35	7.16 ± 1.58*	15.68 ± 3.03	9.29 ± 0.98*	9.68 ± 2.36	7.65 ± 0.85*	9.43 ± 3.93	9.94 ± 2.44	3.93 ± 1.76	3.17 ± 1.06*
Liraglutide Group	9.19 ± 1.61	5.89 ± 0.82*	15.80 ± 2.82	8.71 ± 0.99*	9.07 ± 1.81	7.27 ± 0.78*	9.05 ± 4.27	8.23 ± 2.46*	3.71 ± 1.88	2.15 ± 0.71*
t/z	0.428	5.027	-0.198	2.952	1.433	2.347	0.470	3.488	0.612	5.621
p	0.670	0.000	0.844	0.004	0.155	0.021	0.640	0.001	0.542	0.000

*There was a significant difference between the before treatment and after treatment.

There were no significant differences in NIHSS scores and mRS between the two groups before treatment ($P > 0.05$). After treatment, NIHSS scores and mRS in both groups were lower than those before treatment; the liraglutide group was significantly lower than the control group ($P < 0.05$, **Table 3**).

Table 3. Comparison of NIHSS scores and mRS between the two groups before and after treatment

	NIHSS (points)		mRS (points)	
	Before Treatment	After Treatment	Before Treatment	After Treatment
Control Group	9.44 ± 3.38	6.06 ± 2.92*	4.00 ± 0.99	2.16 ± 0.68*
Liraglutide Group	10.00 ± 3.23	4.74 ± 2.68*	4.02 ± 0.82	1.78 ± 0.65*
t/z	-0.846	2.354	-0.117	-2.755
p	0.399	0.021	0.907	0.006

*There was a significant difference between the before treatment and after treatment.

There were no significant differences in the serum levels of TNF-α, IL-8, hs-CRP, IL-6, NO, and ET-1 between the two groups before treatment ($P > 0.05$). After treatment, the levels of serum TNF-α, IL-8, hs-CRP, IL-6, and ET-1 in both groups were lower than those before treatment, and the level of NO was higher than that before treatment. The liraglutide group showed significant changes compared with the control group ($P < 0.05$, **Table 4** and **Table 5**).

Table 4. Comparison of TNF-α, IL-8, hs-CRP, and IL-6 levels before and after treatment in the two groups

	IL-6 (pg/mL)		TNF-α (pg/mL)		IL-8 (pg/mL)		hs-CRP (mg/L)	
	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment	Before Treatment	After Treatment
Control Group	5.14 ± 0.86	3.66 ± 0.96*	13.99 ± 1.10	5.97 ± 1.72*	18.08 ± 1.46	9.41 ± 1.68*	3.70 ± 0.63	1.77 ± 0.64*
Liraglutide Group	5.19 ± 0.69	3.21 ± 0.82*	13.79 ± 1.19	4.04 ± 1.63*	17.70 ± 1.69	8.31 ± 1.66*	3.52 ± 0.61	1.31 ± 0.54*
t/z	-0.319	2.523	0.879	5.747	1.200	3.288	1.422	3.853
p	0.751	0.013	0.381	0.000	0.233	0.001	0.158	0.000

*There was a significant difference between the before treatment and after treatment.

Table 5. Comparison of NO and ET-1 levels before and after treatment in the two groups

	NO (uml/L)		ET-1 (ng/mL)	
	Before Treatment	**After Treatment**	**Before Treatment**	**After Treatment**
Control Group	24.70 ± 4.75	55.62 ± 4.52*	81.08 ± 5.14	54.58 ± 5.29*
Liraglutide Group	25.26 ± 3.87	57.70 ± 4.48*	80.74 ± 5.50	51.28 ± 4.89*
t	-0.646	-2.311	0.320	3.238
p	0.520	0.023	0.750	0.002

*There was a significant difference between the before treatment and after treatment.

Fisher's exact probability test showed that there were no significant differences in the incidence of adverse reactions such as nausea and vomiting, dizziness and headache, and hypoglycemia between the experimental group and the control group ($P > 0.05$), as shown in **Table 6** below.

Table 6. Comparison of the incidence of adverse reactions between the two groups

	Nausea and vomiting	**Dizziness and headache**	**Hypoglycemia**	**Total incidence**
Control Group	0 (0%)	0 (0%)	4 (8%)	4 (8%)
Liraglutide Group	2 (4%)	1 (2%)	2 (4%)	5 (10%)
χ^2	/	/	/	/
p	0.495	1.000	0.678	1.000

4. Discussion

GLP-1 is an intestinal hormone-related peptide primarily secreted by L-type cells in the small intestinal mucosa in response to postprandial stimulation of glucose and fats. The endogenous GLP-1 has an extremely short half-life of only 1-2 minutes and is rapidly degraded and inactivated after entering the blood circulation. Liraglutide is a synthetic GLP-1 analog with 97% homology to human endogenous GLP-1, exhibiting multiple physiological effects of GLP-1 and a half-life of up to 13 hours. It is currently primarily used for the treatment of T2DM and can stabilize lower blood glucose. GLP-1 can achieve direct binding with pancreatic islet receptors in the body. Atherosclerosis can be improved by regulating TNF-α, hs-CRP, Adiponectin, IL-6, BNP, and CIMT [1]. GLP-1RAs protect blood vessels by promoting angiogenesis and inhibiting oxidative stress, thereby preventing endothelial dysfunction. They also target multiple atherosclerotic processes associated with endothelial dysfunction, including systemic inflammation, vascular smooth muscle cell proliferation, and plaque formation [2]. GLP-1 receptors are widely distributed in the central nervous system, and liraglutide can freely cross the blood–brain barrier to bind to brain GLP-1 receptors, exerting neuroprotective effects beyond glucose reduction [3,4]. Therefore, the neuroprotective role of liraglutide in acute cerebral infarction has become a research hotspot in recent years.

The results of this study showed that after treatment, the NIHSS and mRS scores in the liraglutide group were significantly lower than those in the control group, and the total clinical effective rate was higher than that in the control group. Guan Jinfan et al. [5] demonstrated in animal experiments that liraglutide can significantly reduce the expression of TNF-α and IL-1β, increased the expression of IL-10 and TGF-β, and simultaneously improved neurological function scores and cerebral infarction volume. The recovery of neurological function after cerebral infarction depends on the reconstruction of cerebral vascular network and effective blood perfusion. Vascular regeneration and remodeling are essential conditions for alleviating nerve injury and promoting the repair of neurological function. Bu Yi et al. [6] demonstrated in animal experiments that liraglutide may exert a neuroprotective effect on DM rats with cerebral infarction by promoting IGFBP3 expression and angiogenesis in brain tissue, which supports the findings of the present study.

In addition to the neurological impairment caused by cerebral ischemia, hypoxia, and energy metabolism disorders,

ACI can also aggravate the neurological damage of patients due to inflammatory response, oxidative stress, apoptosis and other mechanisms. Wupo Wuqie et al.[7] found in a rat model of cerebral ischemia with diabetes that liraglutide can reduce the activity of myeloperoxidase in ischemic brain tissue, down-regulate the expression levels of TNF-α and NF-κB, and exert anti-inflammatory and anti-oxidative stress neuroprotective effects in the brain tissue of ischemic rats. In patients with ACI, serum levels of inflammatory factors such as pro-BDNF, IL-6, CRP, and TNF-α are increased, and the levels of pro-BDNF, IL-6, CRP and TNF-α are closely related to the prognosis of ACI patients. The combination of pro-BDNF, IL-6, CRP, and TNF-α for predicting the prognosis of ACI patients has high clinical efficacy [8]. Studies have shown that serum IL-6, TNF-α, hs-CRP, and MMP-9 play significant roles in the occurrence and development of ACI, and are more closely related to the pathogenesis of LAA and SAO [9]. Multiple studies have demonstrated that liraglutide can increase peripheral blood NO levels and reduce ET-1 concentrations in newly diagnosed T2DM patients [10]. Moreover, combined with insulin-intensive treatment of T2DM can promote clinical efficacy, reduce the levels of IL-6, TNF-α, and ET-1, increase the level of NO, enhance blood control, alleviate inflammatory response, and have no significant adverse reactions during treatment [11]. This study selected patients with LAA-type ACI as the research subjects and proved that after treatment, the serum levels of TNF-α, IL-8, hs-CRP and IL-6 in the liraglutide group were significantly lower than those in the control group. Liraglutide also reduces the level of ET-1 and increases the level of NO, with more significant effects than the control group, which is consistent with previous studies. This study found that the experimental group exhibited certain efficacy in improving blood lipid profiles. The evidence was based on the absence of significant differences in LDL-C (low-density lipoprotein cholesterol), TG (triglycerides), TC (total cholesterol), and HDL-C (high-density lipoprotein cholesterol) levels between the two groups before treatment ($p > 0.05$), followed by significant differences after treatment ($p < 0.05$). Specifically, LDL-C, TG, and TC levels decreased compared to the control group post-treatment, while HDL-C levels were higher than those in the control group. These findings warrant further investigation.

5. Conclusion

In conclusion, liraglutide can enhance blood glucose control, alleviate inflammatory response, improve vascular endothelial function, and thus promote neurological prognosis in patients with ACI complicated with T2DM.

Disclosure statement

The authors declare no conflict of interest.

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