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RESEARCH ARTICLE **OPEN ACCESS**

Liraglutide in Obesity Management: Evidence of Weight and Lipid Improvements in a UAE Cohort

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Abstract:

Introduction: Liraglutide, a Glucagon-Like Peptide-1 Receptor Agonist (GLP-1RA), is used for weight management and glycaemic control in obesity. This study aimed to evaluate the effect of liraglutide on weight, glycosylated Hemoglobin (HbA1c), and Low-Density Lipoprotein Cholesterol (LDL-C) over 12 months in a real-world clinical setting.

Methods: This retrospective single-arm longitudinal study included 209 patients with obesity, primarily UAE nationals (86.1%) and females (82.8%). Clinical parameters (weight, HbA1c, and LDL-C) were assessed at baseline, 6 months, and 12 months. Paired-sample t-tests were used to compare clinical parameters at baseline with those at 6 months and 12 months. Subgroup analyses examined sex differences and associations between weight status and HbA1c levels. A p-value < 0.05 was considered significant.

Results: Mean weight decreased from 98.42 ± 16.97 kg at baseline to 92.90 ± 16.83 kg at 6 months ($p<0.001$) and 91.69 ± 16.23 kg at 12 months ($p<0.001$), reflecting a 6.8% reduction. HbA1c declined modestly from 6.33 ± 1.42% to 6.11 ± 1.30% ($p=0.039$). LDL-C decreased from 2.91 ± 0.98 mmol/L to 2.61 ± 0.94 mmol/L ($p=0.009$). Baseline sex differences in weight ($p=0.002$) diminished over time.

Discussion: Liraglutide achieved significant weight and LDL-C reductions and modest glycaemic improvements in a predominantly non-diabetic obese cohort. These outcomes align with previous studies but may vary with dose, duration, and population characteristics.

Conclusion: Liraglutide effectively reduced weight and improved LDL-C levels in young, predominantly female, non-diabetic obese patients from UAE, supporting its role as an adjunct to lifestyle interventions in similar populations.

Keywords: Obesity, Lipid profile, Liraglutide, Glycosylated haemoglobin, Weight reduction.

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1. INTRODUCTION

Obesity is a major global public health concern, affecting over 650 million adults worldwide [1]. It is strongly associated with an increased risk of comorbidities such as hypertension, dyslipidemia, Type 2 Diabetes Mellitus (T2DM), and coronary artery disease. First-line management typically involves lifestyle interventions, including dietary modification, physical activity, and behavioral therapy. However, for many individuals, these strategies alone are insufficient to achieve clinically meaningful and sustained weight loss, necessitating the use of pharmacological agents.

Liraglutide, a Glucagon-Like Peptide-1 (GLP-1) receptor agonist, has demonstrated efficacy as a pharmacologic option for weight management [2]. It enhances glucose-dependent insulin secretion, even before a significant rise in blood glucose levels occurs [3, 4]. While liraglutide is widely used among obese individuals with T2DM, its potential benefits in predominantly non-diabetic populations have also been explored. For instance, a 12-week study conducted in non-diabetic Taiwanese patients showed weight reduction in 5.6% and 6.4% of participants receiving 1.2 mg and 0.6 mg of liraglutide, respectively (both $p < 0.001$). However, the difference between the two doses was not statistically significant (absolute difference −0.8%, 95% CI −0.12 to 0.11) [5]. Similarly, a study involving Indian adolescents reported a mean weight loss of −6.5 ± 4.2 kg and a BMI reduction of −2.35 ± 1.30 kg/m^2, accompanied by improvements in liver enzymes, systolic blood pressure, and triglyceride levels, but no significant change in cholesterol [6].

At a daily dose of 3.0 mg, liraglutide has been shown to facilitate long-term weight loss and improve metabolic parameters, particularly when used in conjunction with dietary and lifestyle modifications [7]. Its mechanisms of action extend beyond glycemic regulation and include delayed gastric emptying, increased satiety, and appetite suppression *via* central nervous system pathways [2, 3]. These mechanisms are particularly relevant in non-diabetic individuals or those with early metabolic dysfunction, where glycemic control may not be the primary therapeutic objective.

Given the high and rising prevalence of obesity in the Middle East, particularly in the United Arab Emirates (UAE), and the limited availability of region-specific data on pharmacologic obesity treatments, evaluating the real-world usefulness of liraglutide in this context is of substantial public health relevance [8].

Therefore, the primary aim of this study was to evaluate the mean percentage reduction in body weight and Hemoglobin A1c (HbA1c) in a cohort of predominantly non-diabetic adults with obesity who received liraglutide treatment in a tertiary care center in the UAE. A secondary objective was to assess changes in lipid parameters and examine subgroup trends based on demographic and clinical characteristics.

2. METHODS

2.1. Study Design and Setting

This study was a retrospective, single-arm longitudinal analysis conducted at a tertiary care hospital in the United Arab Emirates between January 2022 and January 2023. It aimed to assess the impact of liraglutide therapy on body weight, glycosylated hemoglobin (HbA1c), and Low-Density Lipoprotein Cholesterol (LDL-C) over a 12-month follow-up period in a predominantly non-diabetic population with obesity. The study was approved by the Ministry of Health and Prevention (MOHAP) Research Ethics Committee with Approval No. MOHAP/REC/2025/51-2025-F-M. Written informed consents were obtained from all participants, and the study adhered to the guidelines of the Declaration of Helsinki.

2.2. Study Population

A consecutive sampling approach was employed, including all patients who were initiated on liraglutide for weight management during the study period, as identified from electronic medical records. A total of 209 participants were included, the majority of whom were UAE nationals (86.1%) and female (82.8%). Sex was recorded as male or female as self-reported in medical records. The Sex and Gender Equity in Research (SAGER) Guidelines were followed by the authors. Liraglutide treatment began at a starting dose of 0.6 mg/day and was titrated up to 3.0 mg/day based on clinical response and patient tolerance. Although the study primarily targeted non-diabetic individuals, a small number of participants with elevated HbA1c levels were included based on clinical judgment.

2.3. Inclusion and Exclusion Criteria

Participants were eligible for inclusion if they were 18 years or older, had a Body Mass Index (BMI) greater than 30 kg/m^2 (or >27 kg/m^2 for individuals of Asian origin), and had completed at least 12 months of follow-up after initiating liraglutide therapy for weight management. Exclusion criteria included the use of other pharmacological weight-loss therapies during the study period, a history of bariatric surgery, or the presence of significant endocrine disorders, active malignancy, or severe hepatic or renal impairment. Pregnant or breastfeeding women were also excluded due to safety considerations.

2.4. Data Collection and Measurements

Baseline demographic data, including age, sex, and nationality, were collected. Clinical parameters including weight (kg), HbA1c (%), and LDL-C (mmol/L) were measured at baseline, 6 months, and 12 months. BMI was calculated using the standard formula, which is weight in kilograms divided by the square of height in meters (kg/m^2). HbA1c was measured using high-performance liquid chromatography following an overnight fast. LDL-C was either measured directly or estimated using the Friedewald equation where applicable.

2.5. Subgroup Stratification

Participants were stratified into subgroups based on age (<50 years vs. ≥50 years), sex (male vs. female), baseline weight (<100 kg vs. ≥100 kg), and HbA1c levels (<7% vs. ≥7%). These subgroup analyses aimed to examine trends in weight and glycemic parameters across demographic and clinical categories.

2.6. Endpoints

The primary endpoints were changes in body weight at 6 and 12 months, and changes in HbA1c and LDL-C from baseline to 12 months. Secondary analyses explored associations between baseline weight and HbA1c improvement, as well as sex-related differences in weight trends. The proportion of participants achieving HbA1c <7% across weight categories was also evaluated. Data completeness varied across outcomes. A total of 209 participants had complete weight data at 6 months and 181 at 12 months. For HbA1c, 193 participants had paired baseline and 12-month measurements, while 61 participants had complete LDL-C data at both baseline and 12 months. Analyses for each outcome were conducted using the available complete cases, including only participants with paired measurements for the respective variables.

2.7. Statistical Analysis

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS), Version 26 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ± Standard Deviation (SD), and categorical variables as frequencies and percentages. Paired-sample t-tests were used to assess within-group changes in weight, HbA1c, and LDL-C over time. Chi-square tests were employed to evaluate differences in categorical variables between subgroups. Pearson's correlation coefficient was used to examine the relationship between changes in weight and HbA1c. Analyses for each outcome were performed using complete-case analysis, including only participants with available paired measurements for the respective variables. A p-value < 0.05 was considered statistically significant.

3. RESULTS

3.1. Demographic Characteristics

Table **1** presents the baseline demographic and clinical characteristics of the 209 participants. The majority of participants were UAE nationals (86.1%), and females constituted a significantly larger proportion of the sample (82.8%), compared to males (17.2%). Most participants (77.5%) were under the age of 50, indicating a relatively younger population. Regarding body weight, 55.0% of participants weighed less than 100 kg, while 45.0% weighed 100 kg or more. Other participant characteristics are summarized in Table **1**.

3.2. Descriptive Statistics Over Time

Table **2** summarizes the descriptive statistics for key clinical parameters across the study period. Participants' ages ranged from 14 to 70 years, with a mean of 42.25 years. Baseline HbA1c decreased slightly from 6.33% to 6.11% at 12 months (3.5% reduction). LDL cholesterol levels improved over time, declining from a mean of 2.91 mmol/L at baseline to 2.61 mmol/L at 12 months, representing a 10.3% reduction. A substantial decrease in body weight was also observed, with the mean weight dropping from 98.42 kg at baseline to 92.90 kg at 6 months and further to 91.69 kg at 12 months, reflecting a 6.8% total reduction over the study period.

Table 1. Characteristics of the participants.

Parameter		Frequency	%
Nationality	UAE	180	86.1%
	Other	29	13.9%
Sex	F	173	82.8
	M	36	17.2
Age	<50 y	162	77.5
	≥50 y	46	22.0
HbA1c on Presentation	<7%	165	78.9
	≥7%	28	13.4
Weight on Presentation	<100 kg	115	55.0
	≥100 kg	94	45.0

Table 2. Descriptive statistics.

	Minimum	Maximum	Mean	Std. Deviation
Age (y)	14	70	42.25	11.79
HbA1c at baseline (%)	3.65	11.80	6.33	1.42
HbA1c 12 months (%)	4.20	12.20	6.11	1.28
LDL at baseline (mmol/L)	0.18	6.08	2.91	0.98
LDL 12 months (mmol/L)	1.08	6.10	2.61	0.94
Weight at baseline (kg)	64.0	160.0	98.42	16.97
Weight at 6 months (kg)	61.0	150.0	92.9	16.8
Weight at 12 months (kg)	63.0	143.7	91.7	16.2

3.3. Sex Differences in Age, HbA1c, and Weight

Table **3** examines sex-based differences in age, HbA1c, and weight status. Age distribution and baseline HbA1c levels were not significantly different between females and males (p = 0.901 and p = 0.580, respectively). However, significant sex differences were observed in weight status at baseline, with a higher proportion of males weighing ≥100 kg compared to females (p = 0.002). This trend persisted at 6 months (p < 0.001), but by 12 months, the sex difference in weight distribution was no longer statistically significant (p = 0.705).

3.4. Weight Reduction Over Time

Table **4** shows the results of paired-sample t-tests assessing weight changes. The mean weight decreased significantly from 98.42 kg at baseline to 92.90 kg at 6 months, yielding a mean reduction of 5.52 kg (5.6%) (p < 0.001). This downward trend continued, with a further

decrease to 91.69 kg at 12 months, representing a total mean reduction of 6.73 kg (6.8%) from baseline ($p < 0.001$).

Table 3. Sex differences in age, HbA1c, and weight across time points.

Parameter		Female	Male	*P*-value
Age groups	<50 y	135	28	0.901
	≥50 y	38	8	
HbA1c at baseline	<7%	137	28	0.580
	≥7%	25	3	
Weight at baseline	<100 kg	104	11	0.002
	≥100 kg	69	25	
Weight at 6 months	<100 kg	131	13	<0.001
	≥100 kg	42	23	
Weight at 12 months	<100 kg	103	22	0.705
	≥100 kg	48	8	

Table 4. Paired sample t-test analysis of weight reduction over time.

Time Point	Mean (kg)	SD (kg)	Mean (%) Diff. (kg)	SD (kg)	95% CI (kg)	*t*	*p*-value	Cohen's d
Baseline	98.42	16.97						
6 Months	92.90	16.83	5.52 (5.6%)	5.07	4.83–6.22	15.76	<0.001	5.08
12 Months	91.69	16.23	6.73 (6.8%)	6.70	3.97–6.88	7.43	<0.001	5.07

3.5. HbA1c and LDL Changes Over 12 Months

Table **5** outlines changes in HbA1c and LDL-C levels from baseline to 12 months. A modest but statistically significant reduction in HbA1c was observed, decreasing from 6.33% to 6.11%, with a mean difference of 0.22% (3.5% reduction) ($p = 0.039$). LDL-C levels declined significantly from 2.91 mmol/L to 2.61 mmol/L, with a mean decrease of 0.30 mmol/L (10.3% reduction) ($p = 0.009$).

Table 5. Paired sample analysis of HbA1c and LDL changes over 12 months.

Parameter	Time Point	Mean	Std. Deviation	Mean (%) Diff.	SD	95% CI of difference	*P*-value	Cohen's d
HbA1c (%) (n= 63)	Baseline	6.33	1.42	0.22 (3.5%)	0.82	0.01–0.42	0.039*	0.82
	12 Months	6.11	1.30					
LDL (mmol/L) (n= 61)	Baseline	2.91	0.98	0.30 (10.3%)	0.87	0.08–0.52	0.009*	0.87
	12 Months	2.61	0.94					

4. DISCUSSION

Liraglutide led to significant improvements in weight, LDL cholesterol, and HbA1c levels among predominantly young, female participants from the UAE, with initial sex and weight-related differences diminishing over time. In this study, significant weight reduction was observed over 12 months of intervention, with the most notable decline occurring within the first 6 months. This pattern aligns with previous observational studies conducted among Italian overweight and obese patients with T2DM, which demonstrated that liraglutide (3 mg) administered over 24 weeks resulted in modest but clinically relevant weight losses of −2.0 kg and −2.45 kg, respectively [9, 10]. Notably, these studies highlighted that the weight loss was predominantly attributed to reductions in fat mass (ranging from 0.70% to 1.45%), rather than lean tissue mass, suggesting a selective effect of liraglutide on adiposity while preserving muscle mass by preventing muscle protein breakdown. Further support for liraglutide's targeted impact on fat mass comes from a 16-week randomized controlled trial, which showed that liraglutide (3 mg), when combined with diet and behavioural modifications, significantly reduced body fat percentage compared to placebo (−2.0% vs. −0.4%, p = 0.03) [11]. However, it is essential to note that this favourable effect on fat mass percentage has not been consistently replicated in all studies, with some trials failing to demonstrate significant changes in body composition [12-14]. These discrepancies may be influenced by variations in study design, population characteristics, treatment duration, and concurrent lifestyle interventions.

Several studies have evaluated the efficacy of liraglutide for weight reduction in non-diabetic individuals with obesity, providing relevant context for interpreting the findings. A retrospective cohort study in Taiwan investigated the effects of low-dose liraglutide (0.6 mg vs. 1.2 mg daily) over 12 weeks in non-diabetic obese patients and found modest but statistically significant weight loss in both groups [5]. However, no significant difference was observed between the two doses, suggesting that low-dose liraglutide may be useful for some individuals, although higher doses or longer durations might be necessary for more substantial effects. In contrast, a prospective cohort study evaluating liraglutide 3 mg daily over 12 weeks in non-diabetic overweight women with coronary microvascular dysfunction reported a significant weight reduction, indicating a more substantial effect with higher dosing [15]. This supports the idea that the magnitude of weight loss may depend on both the dose and duration of liraglutide treatment. Moreover, these findings are supported by the results of a comprehensive systematic review and Bayesian network meta-analysis by Khera *et al.*, which assessed weight loss efficacy across various pharmacologic agents [16]. The review showed that liraglutide was associated with significantly greater odds of achieving more than 5% weight loss compared to placebo, ranking second only to phentermine-topiramate in overall efficacy. Another systematic review by Zhang *et al.* also supports the effectiveness of liraglutide in non-diabetic obese populations, reporting significant mean weight loss (−5.52 kg) and improvements in secondary outcomes such as systolic blood pressure [17].

In addition to the observed weight reduction, HbA1c levels improved significantly over the 12-month period,

indicating a favorable impact on glycaemic control. This aligns with the known pharmacological profile of liraglutide, which exerts multiple physiological effects due to the widespread distribution of GLP-1 receptors throughout the body [18]. Liraglutide effects include enhanced insulin secretion, improved insulin sensitivity, and suppression of glucagon release, all in a glucose-dependent manner, thus minimizing the risk of hypoglycaemia [19]. Furthermore, liraglutide contributes to weight reduction by slowing gastric emptying, promoting satiety, and reducing appetite through its action on the central nervous system [19]. This study found improvements in HbA1c levels of about 3.5% over 12 months, which aligns with previous reports confirming the glycaemic benefits of liraglutide in individuals with obesity and type 2 diabetes [4]. These findings align with a 2022 systematic review and meta-analysis encompassing 12 randomized controlled trials with a total opf 8,249 non-diabetic obese adults. This study confirmed liraglutide's superiority over placebo in promoting weight loss and reducing Body Mass Index (BMI), highlighting its efficacy in improving metabolic outcomes in this population [20]. Additionally, liraglutide has demonstrated beneficial effects on fasting blood glucose and is generally well tolerated, with gastrointestinal symptoms, such as nausea and diarrhea, being the most commonly reported side effects, and no significant risk of hypoglycemia observed [2, 4, 19].

In addition to its effects on weight and glycemic control, liraglutide significantly improved LDL cholesterol levels over the 12-month period. These findings are consistent with studies reporting lipid-lowering effects of liraglutide in both diabetic and non-diabetic populations [21]. While the precise mechanisms remain incompletely understood, GLP-1 receptor activation may indirectly influence lipid metabolism through improvements in insulin sensitivity, weight loss, and reduced hepatic lipogenesis.

CONCLUSION

The study demonstrated that liraglutide is associated with weight reduction and lipid profile improvement in a predominantly non-diabetic, obese population over 12 months. Significant reductions in body weight and LDL cholesterol were observed, alongside modest but meaningful improvements in HbA1c levels, despite most participants having normal baseline glycaemic status. These findings support the role of liraglutide as a valuable adjunct to lifestyle interventions in managing obesity and related metabolic parameters. Further prospective, controlled studies with larger sample sizes and more extended follow-up periods are warranted to confirm these findings. Future research should also investigate the long-term metabolic and cardiovascular effects of liraglutide use in diverse populations, objectively assess changes in body fat composition, and examine the impact of dietary habits, physical activity levels, and medication adherence on treatment outcomes.

LIMITATIONS

This study has several limitations. Its retrospective single-arm design and lack of a control group limit causal inference and increase the potential for selection and information bias. The use of consecutive sampling from a single center may further reduce generalizability. Additionally, key lifestyle factors such as dietary patterns and physical activity levels were not assessed or controlled, which may have influenced treatment outcomes. The absence of objective body composition measurements prevented evaluation of the quality of weight loss (*i.e.*, changes in fat mass versus lean mass). Furthermore, medication adherence was not objectively monitored. The sex distribution was notably imbalanced, with a predominance of female participants, which may limit the generalizability of the findings to male populations. Missing data, particularly for HbA1c and LDL-C at follow-up, may have affected the precision of outcome estimates and limited our ability to perform multivariable regression analyses to adjust for potential confounders. Analyses were conducted using complete-case data, which may introduce bias if the missingness was not random. Given the extent and pattern of missing data, performing a formal sensitivity analysis was not feasible. The relatively small sample size for some subgroup analyses limited statistical power, increasing the risk of failing to detect true associations. Additionally, the multiple subgroup comparisons conducted in this study raise the possibility of false-positive findings (Type I error).

AUTHORS' CONTRIBUTIONS

The authors confirm their contribution to the paper as follows: study conception and design – FA, data collection – MA, FH, AQ, LK, analysis and interpretation of results – AA, writing - original draft preparation – TM, writing - reviewing and editing – HAH. All authors reviewed the results and approved the final version of the manuscript.

LIST OF ABBREVIATIONS

T2DM	=	Type 2 Diabetes Mellitus
GLP-1	=	Glucagon-Like Peptide-1
GLP-1RA	=	Glucagon-Like Peptide-1 Receptor Agonist
HbA1c	=	Glycosylated Hemoglobin
LDL-C	=	Low-Density Lipoprotein Cholesterol

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was approved by the Ministry of Health and Prevention (MOHAP) Research Ethics Committee with Approval No. MOHAP/REC/2025/51-2025-F-M.

HUMAN AND ANIMAL RIGHTS

All procedures performed in studies involving human participants were in accordance with the ethical standards of institutional and/or research committee and with the 1975 Declaration of Helsinki, as revised in 2013.

CONSENT FOR PUBLICATION

Written informed consents were obtained from all participants.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

STANDARDS OF REPORTING

STROBE guidelines were followed.

FUNDING

Declared None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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REFERENCES

[1] Salam MM, Yousuf R, Salam MW, Haque M. Obesity and overweight: A global public health issue. Advances in Human Biology 2023; 13(1): 154-6.
http://dx.doi.org/10.4103/aihb.aihb_106_22

[2] Lupianez-Merly C, Dilmaghani S, Vosoughi K, Camilleri M. Pharmacologic management of obesity-updates on approved medications, indications and risks. Aliment Pharmacol Ther 2024; 59(4): 475-91.
http://dx.doi.org/10.1111/apt.17856

[3] Martins AC, de la Torre BG, Albericio F. Glucagon-like peptide-1 and glucose-dependent insulinotropic polypeptide agonists for the treatment of obesity and diabetes mellitus. Explor Drug Sci 2024; 2(2): 126-43.
http://dx.doi.org/10.37349/eds.2024.00039

[4] Clark L. GLP-1 receptor agonists: A review of glycemic benefits and beyond. JAAPA 2024; 37(4): 1-4.
http://dx.doi.org/10.1097/01.JAA.0001007388.97793.41

[5] Chou CA, Chuang SF. Evaluation of the efficacy of low-dose liraglutide in weight control among Taiwanese non-diabetes patients. J Diabetes Investig 2020; 11(6): 1524-31.
http://dx.doi.org/10.1111/jdi.13314

[6] Kochar IS, Sethi A. Efficacy and safety of liraglutide in Indian adolescents with obesity. Obes Sci Pract 2019; 5(3): 251-7.
http://dx.doi.org/10.1002/osp4.328

[7] Quinapanta Castro NI, Almeida M, Orbea AF, *et al*. Efficacy of Liraglutide for Weight Loss in Overweight and Obese Non-diabetic Adults: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Cureus 2025; 17(4)e82479
http://dx.doi.org/10.7759/cureus.82479

[8] Yusufali A, Bazargani N, Muhammed K, *et al*. Opportunistic Screening for CVD Risk Factors: The Dubai Shopping for Cardiovascular Risk Study (DISCOVERY). Glob Heart 2015; 10(4): 265-72.
http://dx.doi.org/10.1016/j.gheart.2015.04.008

[9] Perna S, Guido D, Bologna C, Solerte SB, Guerriero F, Isu A, *et al*. Liraglutide and obesity in elderly: efficacy in fat loss and safety in order to prevent sarcopenia. A perspective case series study. Aging Clin Exp Res 2016; 28: 1251-7.
http://dx.doi.org/10.1007/s40520-015-0525-y

[10] Rondanelli M, Perna S, Astrone P, Grugnetti A, Solerte SB, Guido D. Twenty-four-week effects of liraglutide on body composition, adherence to appetite, and lipid profile in overweight and obese patients with type 2 diabetes mellitus. Patient Prefer Adherence 2016; •••: 407-13.
http://dx.doi.org/10.2147/PPA.S97383

[11] Kadouh HC, Halawi H, Chedid V, Khemani D, Burton D, Eckert DJ, *et al*. TU1921-liraglutide treatment modulates regional body fat distribution in individuals with obesity. Gastroenterology 2018; 154(6)S-1054
http://dx.doi.org/10.1016/S0016-5085(18)33527-3

[12] Aladel A, Jamilah A, Basaad R. Effect of Liraglutide on Fat Mass Percentage Among Overweight and Obese Adults with Type 2 Diabetes: A Systematic Review. Diabetes Metab Syndr Obes 2023; 16: 3381-91.
http://dx.doi.org/10.2147/DMSO.S417374

[13] Apperley L, Parkinson J, Senniappan S. Liraglutide Treatment Improves Glycaemic Dysregulation, Body Composition, Cardiometabolic Variables and Uncontrolled Eating Behaviour in Adolescents with Severe Obesity. J Clin Res Pediatr Endocrinol 2024; 10
http://dx.doi.org/10.4274/jcrpe.galenos.2024.2023-10-10

[14] Pandey A, Patel KV, Segar MW, Ayers C, Linge J, Leinhard OD, *et al*. Effect of liraglutide on thigh muscle fat and muscle composition in adults with overweight or obesity: results from a randomized clinical trial. J Cachexia Sarcopenia Muscle 2024; 15(3): 1072-83.
http://dx.doi.org/10.1002/jcsm.13445

[15] Suhrs HE, Raft KF, Bové K, Madsbad S, Holst JJ, Zander M, *et al*. Effect of liraglutide on body weight and microvascular function in non-diabetic overweight women with coronary microvascular dysfunction. Int J Cardiol 2019; 283: 28-34.
http://dx.doi.org/10.1016/j.ijcard.2018.12.005

[16] Khera R, Murad MH, Chandar AK, Dulai PS, Wang Z, Prokop LJ, *et al*. Association of Pharmacological Treatments for Obesity With Weight Loss and Adverse Events: A Systematic Review and Meta-analysis. JAMA 2016; 315(22): 2424-34.
http://dx.doi.org/10.1001/jama.2016.7602

[17] Zhang P, Liu Y, Ren Y, Bai J, Zhang G, Cui Y. The efficacy and safety of liraglutide in the obese, non-diabetic individuals: a systematic review and meta-analysis. Afr Health Sci 2019; 19(3): 2591-9.
http://dx.doi.org/10.4314/ahs.v19i3.35

[18] Zheng Z, Zong Y, Ma Y, Tian Y, Pang Y, Zhang C, *et al*. Glucagon-like peptide-1 receptor: mechanisms and advances in therapy. Signal Transduct Target Ther 2024; 9(1): 234.
http://dx.doi.org/10.1038/s41392-024-01931-z

[19] Tilinca MC, Tiuca RA, Burlacu A, Varga AA. 2021 update on the use of liraglutide in the modern treatment of 'diabesity': a narrative review. Medicina (B Aires) 2021; 57(7): 669.
http://dx.doi.org/10.3390/medicina57070669

[20] Barboza JJ, Huamán MR, Melgar B, Diaz-Arocutipa C, Valenzuela-Rodriguez G, Hernandez AV. Efficacy of Liraglutide in Non-Diabetic Obese Adults: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. J Clin Med 2022; 11(11): 2998.
http://dx.doi.org/10.3390/jcm11112998

[21] Alonso-Troncoso I, Carollo-Limeres C, Rios-Prego M, Guler I, Cadarso-Suarez C. F-Mariño A. Liraglutide in a real-world setting: Joint modeling of metabolic response, prediction of efficacy, and cardiovascular risk. Endocrinol Diabetes Nutr 2019; 66(6): 376-84.
http://dx.doi.org/10.1016/j.endinu.2018.09.005