

# 司美格鲁肽注射液治疗不同体质量指数2型糖尿病对糖脂代谢和脂肪因子的影响

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**【摘要】** 目的 分析司美格鲁肽注射液治疗不同体质量指数(BMI)2型糖尿病(T2DM)对糖脂代谢和脂肪因子的影响。方法 选择2022年5月至2024年5月我院收治的T2DM患者143例纳入研究,按照患者的BMI分为正常组31例( $18.5 \text{ kg/m}^2 \leq \text{BMI} < 24 \text{ kg/m}^2$ )、超重组49例( $24 \text{ kg/m}^2 \leq \text{BMI} < 28 \text{ kg/m}^2$ )、肥胖组63例( $\text{BMI} \geq 28 \text{ kg/m}^2$ ),均根据指南接受常规治疗,同时给予司美格鲁肽注射液治疗,3组均治疗12周。比较3组治疗前、治疗12周后糖代谢、胰岛功能、脂代谢、人体成分、脂肪因子和治疗期间安全性。结果 与治疗前比较,治疗12周后3组血清空腹血糖(FPG)、餐后2 h血糖(2hPG)、糖化血红蛋白(HbA1c)、胰岛素抵抗指数(HOMA-IR)、血清甘油三酯(TG)、总胆固醇(TC)及低密度脂蛋白胆固醇(LDL-C)水平降低,且肥胖组低于正常组、超重组,超重组低于正常组( $P < 0.05$ )。与治疗前比较,治疗12周后3组空腹C肽(FCP)、餐后2 h C肽(2hCP)、鸢尾素(irisin)、降脂素(adipsin)、网膜素-1(omentin-1)均升高,且肥胖组高于正常组、超重组,超重组高于正常组呈升高趋势( $P < 0.05$ )。与治疗前比较,治疗12周后3组血清高密度脂蛋白胆固醇(HDL-C)水平均升高( $P < 0.05$ )。治疗前超重组、肥胖组BMI、全身总脂肪质量(WBFM)水平高于正常组,肥胖组高于超重组( $P < 0.05$ );与治疗前比较,治疗12周后超重组、肥胖组BMI、WBFM水平均降低,但肥胖组高于正常组、超重组,超重组高于正常组( $P < 0.05$ )。正常组、超重组、肥胖组治疗期间不良反应发生率依次为12.90%(4/31)、10.20%(5/49)、11.11%(7/63),3组差异无统计学意义( $P > 0.05$ )。结论 司美格鲁肽注射液可改善不同BMI的T2DM患者糖脂代谢、胰岛功能、脂肪因子,调节人体成分,对超重特别是肥胖的T2DM患者糖脂代谢、胰岛功能以及脂肪因子具有较强的改善作用,未增加安全风险。

**【关键词】** 2型糖尿病; 司美格鲁肽注射液; 体质量指数; 糖脂代谢; 脂肪因子; 人体成分

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**【Abstract】** **Objective** To investigate the effects of Semaglutide injection on glycolipid metabolism and adipokine levels in the treatment of type 2 diabetes mellitus (T2DM) patients with varying body mass index (BMI). **Methods** A total of 143 patients with T2DM admitted to our hospital between May 2022 and May 2024 were enrolled in this study. Based on their BMI, the patients were categorized into three groups: the normal-weight group (31 cases,  $18.5 \text{ kg/m}^2 \leq \text{BMI} < 24 \text{ kg/m}^2$ ), the super-recombinant group (49 cases,  $24 \text{ kg/m}^2 \leq \text{BMI} < 28 \text{ kg/m}^2$ ), and the obese group (63 cases,  $28 \text{ kg/m}^2 \leq \text{BMI}$ ). All participants received standard guideline-based conventional treatment and were additionally administered semaglutide injections for a duration of 12 weeks. The study compared glucose metabolism, islet  $\beta$ -cell function, lipid metabolism, body composition, adipokine levels before treatment and at 12 weeks post-treatment, as well as safety profiles during the treatment period across the three groups. **Results** After 12 weeks of treatment, the levels of fasting blood glucose (FPG), 2-hour postprandial blood glucose (2 h PG), glycosylated hemoglobin (HbA1c), insulin resistance index (HOMA-IR), serum triglycerides (TG), total cholesterol (TC), and low-density lipoprotein cholesterol (LDL-C) decreased in all three groups. Specifically, the reductions were more pronounced in the obesity group compared to the normal group and the super-reorganization group, and the super-reorganization group showed intermediate reductions between the obesity

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and normal groups ( $P < 0.05$ ). Conversely, fasting C-peptide (FCP), 2-hour postprandial C-peptide (2 h CP), irisin, adiponin, and omentin-1 levels increased in all three groups after treatment, with the greatest increases observed in the obesity group, followed by the super-reorganization group, both of which were significantly higher than the normal group ( $P < 0.05$ ). Additionally, high-density lipoprotein cholesterol (HDL-C) levels increased in all three groups after 12 weeks of treatment ( $P < 0.05$ ). Baseline BMI and total body fat mass (WBFM) were significantly higher in the obesity group compared to the super-reorganization group, which in turn were higher than the normal group ( $P < 0.05$ ). After treatment, BMI and WBFM decreased in both the obesity and super-reorganization groups, but remained significantly higher than in the normal group ( $P < 0.05$ ). The incidence of adverse reactions during treatment was 12.90% (4/31), 10.20% (5/49), and 11.11% (7/63) in the normal, super-reorganization, and obesity groups, respectively, with no statistically significant differences among the groups ( $P > 0.05$ ). **Conclusion** Smiglutide injection can improve glucose and lipid metabolism, islet function, and adipokine levels in T2DM patients across different BMI categories, while also regulating body composition. It demonstrates a particularly strong improvement effect on glucose and lipid metabolism, islet function, and adipokine levels in overweight and obese T2DM patients. Additionally, smiglutide does not increase safety risks.

**【Key words】** type 2 diabetes mellitus; semaglutide injection; body mass index; glycolipid metabolism; lipofactor; body composition

2型糖尿病(T2DM)为临床常见内分泌疾病,中国是世界上糖尿病患者最多的国家,且其发病率在我国呈上升趋势,已成为严重公共卫生问题<sup>[1-2]</sup>。生活方式干预是T2DM的基础治疗措施,同时其治疗也是综合性的,包括降压、降糖、调脂、抗血小板等<sup>[3]</sup>。司美格鲁肽注射液为长效胰高糖素样肽-1受体激动剂,可调节血糖代谢,改变机体食物/奖励系统,具有良好的降糖疗效和安全性,作为T2DM的治疗药物引入国内,同时近年来也在肥胖症中具有良好应用<sup>[4-5]</sup>。研究<sup>[6]</sup>指出,超重、肥胖人群发生T2DM的风险更高,但仍存在部分T2DM患者体型正常,不同体质量指数(BMI)T2DM患者接受司美格鲁肽注射液治疗时可能表现出不同结果。基于此,本研究将司美格鲁肽注射液应用于不同BMI的T2DM患者的治疗,分析患者糖脂代谢和脂肪因子变化,以期为临床提供参考。

## 1 资料与方法

**1.1 一般资料** 选择2022年5月至2024年5月我院收治的T2DM患者143例纳入研究,按照患者的BMI分为正常组31例( $18.5 \text{ kg/m}^2 \leq \text{BMI} < 24 \text{ kg/m}^2$ )、

超重组49例( $24 \text{ kg/m}^2 \leq \text{BMI} < 28 \text{ kg/m}^2$ )、肥胖组63例( $\text{BMI} \geq 28 \text{ kg/m}^2$ )<sup>[7]</sup>。纳入标准:T2DM符合指南<sup>[8]</sup>中相关标准者;在饮食控制和运动基础上,口服二甲双胍血糖控制不理想者;年龄超过18岁者;重要脏器功能正常者;患者签署知情同意书者等。排除标准:甲状腺癌患者或有家族史者;合并抑郁倾向者;难以配合治疗者;合并严重感染者;合并恶病质者;因其他内分泌疾病导致的肥胖者;其他类型糖尿病如1型糖尿病、妊娠糖尿病者等。剔除标准:不耐受治疗者;需接受手术或其他类型治疗者;撤回知情同意者;未完成全程治疗者。3组一般资料差异有统计学意义( $P > 0.05$ ),见表1。临床试验经我院医学伦理委员会审核批准(伦理审批号:P-SL-2021168)。

**1.2 治疗方法** 所有患者均根据指南进行治疗,在改善生活方式(控制饮食、增加锻炼)的基础上应用降压、降糖、调脂、抗血小板等药物治疗,在此基础上,3组患者均接受司美格鲁肽注射液(国药准字SJ20240023, Novo Nordisk A/S, 1.34 mg/mL, 1.5 mL)治疗,皮下注射0.25 mg/次,1次/周,4周后调整至每次0.5 mg/次,监测血糖、体质量变化,据

表1 3组一般资料比较

Tab.1 Comparison of general data in three groups

$\bar{x} \pm s$

组别	例数	年龄( $\bar{x} \pm s$ )/岁	性别[例(%)]		T2DM病程( $\bar{x} \pm s$ )/年	吸烟[例(%)]	饮酒[例(%)]
			男	女			
正常组	31	55.89 ± 5.72	17(54.84)	14(45.16)	11.88 ± 2.31	7(22.58)	5(16.13)
超重组	49	56.11 ± 5.05	26(53.06)	23(46.94)	11.73 ± 2.22	11(22.45)	8(16.33)
肥胖组	63	55.75 ± 5.43	39(61.90)	24(38.10)	12.01 ± 2.42	16(25.40)	11(17.46)
$F/\chi^2$ 值		0.062	0.983		0.199	0.163	0.037
$P$ 值		0.940	0.612		0.820	0.922	0.981

此调节药量,但不应高于1 mg/次,治疗周期为12周。

### 1.3 观察指标

**1.3.1 糖代谢** 取空腹静脉、餐后2 h血液标本3 mL并制备血清(3 500 r/min转速下离心10 min),采用己糖激酶法检测空腹血糖(FPG)、餐后2 h血糖(2hPG),试剂盒来自上海高踪医疗器械科技有限公司;通过免疫荧光层析法检测血清糖化血红蛋白(HbA1c)水平(江西达优医疗科技有限公司)。治疗前、治疗12周后检测。

**1.3.2 胰岛功能** 同1.3.1采集血液标本并制备血清,通过胶乳免疫比浊法检测血清胰岛素(FINS)水平(广州万孚生物技术股份有限公司),计算胰岛素抵抗指数(HOMA-IR)=FPG×FINS/22.5,通过化学发光免疫分析法检测空腹C肽(FCP)、餐后2 h C肽(2hCP)水平(北京福瑞润康生物技术有限公司)。治疗前、治疗12周后检测。

**1.3.3 脂代谢** 同1.3.1采集血液标本,通过HMS200型全自动生化分析仪(常德普施康生物科技有限公司)检测血清甘油三酯(TG)、高密度脂蛋白胆固醇(HDL-C)、总胆固醇(TC)及低密度脂蛋白胆固醇(LDL-C)水平。治疗前、治疗12周后

检测。

**1.3.4 人体成分** 记录3组BMI,通过深圳市乐福衡器有限公司提供的CF366型人体脂肪测量仪检测全身总脂肪质量(WBFM)、全身总瘦组织质量(FFM)。治疗前、治疗12周后检测。

**1.3.5 脂肪因子** 同1.3.1采集血液标本、制备血清标本,通过酶联免疫吸附试验检测血清鸢尾素(irisin)、降脂素(adipsin)、网膜素-1(omentin-1)水平(美国B&D公司)。治疗前、治疗12周后检测。

**1.3.6 安全性** 比较3组治疗期间不良反应发生情况,包括恶心呕吐、腹泻、便秘、轻度低血糖等。

**1.4 统计学方法** 使用SPSS 26.0统计软件进行分析。计数资料以[例(%)]表示,比较用 $\chi^2$ 检验。符合正态分布的计量资料表示为均数±标准差,多组间比较采用单因素方差分析,进一步两两组间比较采用LSD-*t*检验。以 $P < 0.05$ 为差异有统计学意义。

## 2 结果

**2.1 治疗前、治疗12周后3组糖代谢比较** 与治疗前比较,治疗12周后3组血清FPG、2hPG、HbA1c水平降低,且肥胖组低于正常组、超重组,超重组低于正常组( $P < 0.05$ )。见表2。

表2 治疗前、治疗12周后3组糖代谢比较

Tab.2 Comparison of glucose metabolism in three groups before treatment and 12 weeks after treatment		$\bar{x} \pm s$			
时间	组别	例数	FPG/(mmol/L)	2hPG/(mmol/L)	HbA1c/%
治疗前	正常组	31	8.41 ± 1.42	13.94 ± 1.19	8.14 ± 1.19
	超重组	49	8.63 ± 1.28	14.11 ± 1.15	8.09 ± 1.25
	肥胖组	63	8.71 ± 1.34	14.15 ± 1.52	8.21 ± 1.11
	<i>F</i> 值		0.526	0.265	0.146
	<i>P</i> 值		0.592	0.768	0.864
治疗12周后	正常组	31	7.46 ± 0.35*	8.41 ± 0.54*	7.01 ± 0.24*
	超重组	49	7.29 ± 0.46*#	8.07 ± 0.51*#	6.87 ± 0.38*#
	肥胖组	63	6.80 ± 0.48*#&	7.20 ± 0.58*#&	6.64 ± 0.33*#&
	<i>F</i> 值		28.480	62.472	14.625
	<i>P</i> 值		< 0.001	< 0.001	< 0.001

注:与治疗前比较,\* $P < 0.05$ ;与正常组比较,# $P < 0.05$ ;与超重组比较,& $P < 0.05$

**2.2 治疗前、治疗12周后3组胰岛功能比较** 与治疗前比较,治疗12周后3组FCP、2hCP均升高,且肥胖组高于正常组、超重组,超重组高于正常组( $P < 0.05$ );治疗12周后3组HOMA-IR水平降低,且肥胖组低于正常组、超重组,超重组低于正常组( $P < 0.05$ )。见表3。

**2.3 治疗前、治疗12周后3组脂代谢比较** 与治疗前比较,治疗12周后3组血清TG、TC、LDL-C水平均降低,且肥胖组低于正常组、超重组,超重组低

于正常组( $P < 0.05$ );治疗12周后3组血清HDL-C水平均升高( $P < 0.05$ )。见表4。

**2.4 3组人体成分比较** 治疗前正常组、超重组、肥胖组BMI、WBFM水平呈升高趋势( $P < 0.05$ );治疗12周后超重组、肥胖组BMI、WBFM水平均降低,但肥胖组高于正常组、超重组,超重组高于正常组BMI、WBFM水平仍呈升高趋势( $P < 0.05$ )。见表5。

**2.5 3组脂肪因子比较** 治疗12周后3组血清

表3 治疗前、治疗12周后3组胰岛功能比较

Tab.3 Comparison of islet function in three groups before treatment and 12 weeks after treatment  $\bar{x} \pm s$

时间	组别	例数	HOMA-IR	FCP/(ng/mL)	2 h CP/(ng/mL)
治疗前	正常组	31	3.41 ± 0.51	1.56 ± 0.41	5.14 ± 1.05
	超重组	49	3.45 ± 0.57	1.55 ± 0.42	5.08 ± 1.02
	肥胖组	63	3.51 ± 0.47	1.48 ± 0.43	4.92 ± 1.11
	F值		0.436	0.544	0.550
	P值		0.647	0.581	0.578
治疗12周后	正常组	31	2.67 ± 0.26*	3.82 ± 0.56*	9.11 ± 0.77*
	超重组	49	2.42 ± 0.39*#	4.32 ± 0.44*#	9.86 ± 0.86*#
	肥胖组	63	2.03 ± 0.35*#&	5.04 ± 0.63*#&	11.80 ± 0.81*#&
	F值		39.465	55.236	138.596
	P值		< 0.001	< 0.001	< 0.001

注:与治疗前比较,\* $P < 0.05$ ;与正常组比较,# $P < 0.05$ ;与超重组比较,& $P < 0.05$

表4 治疗前、治疗12周后3组脂代谢比较

Tab.4 Comparison of lipid metabolism in three groups before treatment and 12 weeks after treatment  $(\bar{x} \pm s)/(mmol/L)$

时间	组别	例数	TG	HDL-C	TC	LDL-C
治疗前	正常组	31	3.06 ± 0.87	1.16 ± 0.32	5.49 ± 0.45	4.50 ± 0.81
	超重组	49	3.10 ± 0.85	1.14 ± 0.34	5.54 ± 0.86	4.52 ± 0.83
	肥胖组	63	3.18 ± 0.82	1.11 ± 0.32	5.43 ± 0.23	4.63 ± 0.82
	F值		0.249	0.270	0.526	0.368
	P值		0.780	0.763	0.592	0.693
治疗12周后	正常组	31	2.75 ± 0.21*	1.33 ± 0.33*	4.92 ± 0.35*	4.01 ± 0.44*
	超重组	49	2.53 ± 0.25*#	1.36 ± 0.36*	4.54 ± 0.31*#	3.90 ± 0.41*#
	肥胖组	63	2.17 ± 0.26*#&	1.40 ± 0.34*	4.14 ± 0.33*#&	3.55 ± 0.42*#
	F值		65.001	0.467	61.889	15.975
	P值		< 0.001	0.628	< 0.001	< 0.001

注:与治疗前比较,\* $P < 0.05$ ;与正常组比较,# $P < 0.05$ ;与超重组比较,& $P < 0.05$

表5 治疗前、治疗12周后3组人体成分比较

Tab.5 Comparison of body composition in three groups before treatment and 12 weeks after treatment  $\bar{x} \pm s$

时间	组别	例数	BMI/(kg/m <sup>2</sup> )	WBFM/kg	FFM/kg
治疗前	正常组	31	22.02 ± 1.73	16.85 ± 1.77	58.84 ± 3.86
	超重组	49	27.17 ± 1.71#	19.09 ± 1.62#	58.02 ± 3.60
	肥胖组	63	31.87 ± 1.60#&	22.11 ± 1.72#&	59.20 ± 3.71
	F值		375.084	108.988	1.419
	P值		< 0.001	< 0.001	0.245
治疗12周后	正常组	31	21.98 ± 1.61	16.66 ± 1.41	59.05 ± 3.41
	超重组	49	25.65 ± 1.67*#	18.92 ± 1.52*#	59.20 ± 3.58
	肥胖组	63	29.28 ± 1.85*#&	19.73 ± 1.44*#&	59.25 ± 3.06
	F值		190.817	46.136	0.038
	P值		< 0.001	< 0.001	0.963

注:与治疗前比较,\* $P < 0.05$ ;与正常组比较,# $P < 0.05$ ;与超重组比较,& $P < 0.05$

irisin、adipsin、omentin-1水平均升高,且肥胖组高于正常组、超重组,超重组高于正常组( $P < 0.05$ )。见表6。

表6 治疗前、治疗12周后3组脂肪因子比较

Tab.6 Comparison of adipokines in three groups before treatment and 12 weeks after treatment ( $\bar{x} \pm s$ )/(ng/mL)

时间	组别	例数	irisin	adipsin	omentin-1
治疗前	正常组	31	4.56 ± 0.76	3247.86 ± 361.85	52.09 ± 3.20
	超重组	49	4.52 ± 0.87	3224.71 ± 389.72	51.82 ± 3.21
	肥胖组	63	4.46 ± 0.84	3192.46 ± 378.13	51.44 ± 3.05
	F值		0.166	0.244	0.491
	P值		0.847	0.784	0.613
治疗12周后	正常组	31	4.96 ± 0.65*	4729.97 ± 513.31*	57.80 ± 4.14*
	超重组	49	5.33 ± 0.66*#	5145.11 ± 455.24*#	63.13 ± 4.43*#
	肥胖组	63	5.80 ± 0.71*#&	5676.06 ± 421.22*#&	67.10 ± 4.11*#&
	F值		17.122	48.864	50.979
	P值		< 0.001	< 0.001	< 0.001

注:与治疗前比较,\* $P < 0.05$ ;与正常组比较,# $P < 0.05$ ;与超重组比较,& $P < 0.05$

2.6 3组安全性比较 正常组、超重组、肥胖组治疗期间不良反应发生率比较,12.90%(4/31)、10.20%(5/49)、11.11%(7/63),差异无统计意义( $P > 0.05$ )。见表7。

表7 治疗期间3组安全性比较

Tab.7 Comparison of safety in three groups during treatment

例(%)

组别	例数	恶心呕吐	腹泻	便秘	轻度低血糖	合计
正常组	31	1(3.23)	1(3.23)	0(0.00)	2(6.45)	4(12.90)
超重组	49	2(4.08)	1(2.04)	2(4.08)	0(0.00)	5(10.20)
肥胖组	63	2(3.17)	2(3.17)	3(4.76)	0(0.00)	7(11.11)
$\chi^2$ 值						0.140
P值						0.932

### 3 讨论

糖尿病以高血糖为特征,其中主要类型为T2DM,随着病情进展可损害视网膜、心脏、肾等组织器官,产生多种并发症,威胁患者生命安全健康<sup>[9-11]</sup>。超重或肥胖为慢性代谢性疾病,可增加T2DM的发生风险,但仍存在BMI正常的T2DM患者,探索不同BMI的T2DM患者最佳治疗方式很有必要<sup>[12-13]</sup>。司美格鲁肽注射液是兼顾强效降糖及代谢调节的制剂,本研究将其应用于不同BMI的T2DM的临床治疗,取得一定成果。

T2DM患者发病特征为相对胰岛素缺乏,机体存在胰岛素抵抗,可降低FCP、2hCP水平,提高HOMA-IR值,同时糖脂代谢紊乱,相关指标存在异常升高或降低,irisin、adipsin、omentin-1为机体脂肪因子,具有调节糖脂代谢的作用,T2DM患者脂肪因子分泌也存在降低<sup>[14-16]</sup>。本研究中,治疗12周后,超重组、肥胖组血清FPG、2hPG、HbA1c、HOMA-IR、TG、TC、LDL-C水平低于正常组,肥胖组低于超重组,超重组、肥胖组血清FCP、餐后2h

CP、irisin、adipsin、omentin-1高于正常组,肥胖组高于超重组,治疗12周后3组血清HDL-C水平均升高,但组间比较差异无统计学意义( $P > 0.05$ ),提示司美格鲁肽注射液可改善不同BMI的T2DM患者糖脂代谢、胰岛功能、脂肪因子分泌,对超重特别是肥胖的T2DM患者糖脂代谢、胰岛功能以及脂肪因子具有较强的改善作用。司美格鲁肽注射液能通过刺激胰岛素分泌和降低胰高血糖素分泌来改善胰岛细胞功能,降低血糖水平<sup>[17-18]</sup>;其还可促使高脂肪组织、肌肉对血糖进行利用,调节脂质转运、增加脂肪酸 $\beta$ 氧化,减轻与脂肪增生相关的炎症反应,调节脂肪组织中细胞因子分泌,进而影响脂肪子irisin、adipsin、omentin-1分泌,并控制肝脏糖异生作用,游离脂肪酸的水平,从而改善胰岛素抵抗,共同调节糖脂代谢<sup>[19-20]</sup>。此外研究<sup>[21]</sup>指出,司美格鲁肽注射液降糖和减重的双重作用能够针对超重、肥胖T2DM患者的病理生理特点,降糖和减重效果更好,也更持久,与本研究结果类似。王若等<sup>[22]</sup>研究亦指出,司美格鲁肽注射液能调节超重T2DM患者脂肪因子代谢,与本研究结果相互印证。

司美格鲁肽注射液能通过作用于下丘脑,发挥延缓胃排空和减少食欲的作用,有助于降低餐后血液循环中血糖升高的速度,避免血糖转换为脂肪贮存,有助于减轻体重<sup>[23-24]</sup>。研究<sup>[25]</sup>指出,司美格鲁肽可使连续3餐随意进餐的能量摄入降低18%~35%,控制能量摄入进而降低患者肥胖情况及机体组织中脂肪含量。而司美格鲁肽注射液可通过调节脂质和有机酸代谢、减少脂肪积累以及促进肌肉蛋白质合成等,提高组织代谢,抑制脂肪的积累,进而调节T2DM病情进展<sup>[26-27]</sup>。本研究中,治疗前超重组、肥胖组BMI、WBFM水平高于正常组,肥胖组高于超重组,与治疗前比较,治疗12周后3组BMI、WBFM水平均降低,但超重组、肥胖组仍高于正常组,肥胖组高于超重组,3组治疗期间不良反应发生率接近,提示司美格鲁肽注射液可调节超重、肥胖T2DM患者人体成分,且未增加安全风险。

综上所述,司美格鲁肽注射液可改善不同BMI T2DM患者糖脂代谢、胰岛功能、脂肪因子,调节人体成分,对超重特别是肥胖的T2DM患者糖脂代谢、胰岛功能以及脂肪因子具有较强的改善作用,未增加安全风险。但本研究存在纳入不同BMI的T2DM患者均来自本院、病例数有限、未进行长期随访,可进一步探究司美格鲁肽注射液在不同BMI T2DM患者中的应用效果,并分析其治疗机制,为改善不同BMI T2DM患者病情提供依据。

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