

·临床研究·

儿童及青少年颅咽管瘤患者术后肥胖及糖脂代谢特征分析

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摘 要:【目的】探讨儿童及青少年颅咽管瘤患者术后超重/肥胖的发生情况、糖脂代谢特征及其影响因素。【方法】回顾性分析2006年1月至2024年2月诊断为颅咽管瘤并接受手术治疗且在中山大学孙逸仙纪念医院进行随访管理的44例0-18岁患者的临床资料,分析总结术后超重/肥胖和糖脂代谢特征,及其影响因素。【结果】①共纳入44例患者,手术时平均年龄为(8.57±4.25)岁,末次随访时平均年龄为(16.02±5.36)岁,平均随访时长为(7.45±4.64)年(范围:8个月至17.5年)。②患者术前超重/肥胖的发生率为40.91%(以超重为主,占27.27%);术后第一年超重/肥胖发生率升高至68.18%(以肥胖为主,占50.00%)。③术后44/44例(100%)患者有空腹血糖(FPG)资料,其中2例(4.55%)存在空腹血糖受损(IFG);20/44例患者检测了空腹胰岛素(FINS),有11/20例(55.00%)存在胰岛素抵抗;12/44例患者接受了口服葡萄糖耐量试验(OGTT)/胰岛素释放试验检查,有12/12例(100%)存在胰岛素释放异常、5/12例(41.67%)存在高胰岛素血症、3/12例(25.00%)存在糖尿病(DM)。④术后44/44例(100%)患者有血脂谱资料,36/44例(81.82%)存在脂代谢异常,表现为高非高密度脂蛋白胆固醇(nHDL)血症29例(65.91%)、高甘油三酯(TG)血症29例(65.91%)、低高密度脂蛋白胆固醇(HDL-C)血症22例(50.00%)、混合型高脂血症13例(29.55%)。⑤logistic回归分析发现患者术前超重/肥胖以及术后糖代谢异常是术后超重/肥胖的影响因素。6.术后,与体质量正常的患者相比,超重/肥胖患者的FINS水平(14.22 mU/L vs. 3.57 mU/L)、胰岛素抵抗指数(HOMA-IR)(2.88 vs. 0.52)及异常糖代谢发生率(77.78% vs. 23.08%)更高,而脂代谢异常发生率(94.44% vs. 73.08%)无明显差异。【结论】儿童及青少年颅咽管瘤患者术前超重/肥胖的发生率高于正常儿童,术后更为显著;且存在高发的糖脂代谢紊乱,是代谢综合征的高风险人群,临床工作中应予以重视并定期评估。当患者术后出现肥胖时,尤其要关注其糖代谢异常的发生可能,建议进一步监测其糖稳态指标。

关键词: 儿童; 青少年; 颅咽管瘤; 超重; 肥胖; 糖脂代谢

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Analysis of Postoperative Obesity and Glucolipid Metabolism Characteristics in Children and Adolescent with Craniopharyngioma

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Abstract:【Objective】To investigate the incidence, glucolipid metabolism characteristics, and associated factors of postoperative overweight/obesity in children and adolescent with craniopharyngioma.【Methods】A retrospective study was conducted using follow-up data from 44 patients (aged 0-18 years) diagnosed with craniopharyngioma who underwent

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surgical treatment and received follow-up care at Sun Yat-sen Memorial Hospital, Sun Yat-sen University, between January 2006 and February 2024. The incidence and glucolipid metabolism characteristics of overweight/obesity were summarized, and potential influencing factors were analyzed.【Results】A total of 44 patients were enrolled. The mean age at surgery was (8.57 ± 4.25) years, and the mean age at last follow-up was (16.02 ± 5.36) years. The mean follow-up duration was (7.45 ± 4.64) years (range, 8 months to 17.5 years). The preoperative incidence of overweight/obesity was 40.91% (predominantly overweight, 27.27%), which increased to 68.18% postoperatively (predominantly obesity, 50.00%). Postoperatively, all patients (44/44, 100%) underwent fasting plasma glucose (FPG) testing, among whom 2 (4.55%) had impaired fasting glucose (IFG). Fasting insulin (FINS) was measured in 20 patients, and insulin resistance was identified in 11 (55.00%). Oral glucose tolerance testing (OGTT) with insulin release assessment was performed in 12 patients, and all (100%) exhibited abnormal insulin secretion, including 5 (41.67%) with hyperinsulinemia and 3 (25.00%) diagnosed with diabetes mellitus (DM). All patients (44/44, 100%) underwent lipid profile assessment. The incidence of dyslipidemia was 81.82% (36/44), including elevated non-high-density lipoprotein cholesterol (29/44, 65.91%), hypertriglyceridemia (29/44, 65.91%), low high-density lipoprotein cholesterol (22/44, 50.00%), and mixed hyperlipidemia (13/44, 29.55%). Logistic regression analysis indicated that preoperative overweight/obesity and postoperative abnormal glucose metabolism were significant factors associated with postoperative overweight/obesity. Compared with normal-weight patients, those with postoperative overweight/obesity had higher FINS levels (14.22 mU/L *vs.* 3.57 mU/L), higher insulin resistance index (HOMA-IR) (2.88 *vs.* 0.52), and a higher incidence of abnormal glucose metabolism (77.78% *vs.* 23.08%). However, no significant difference was observed in the incidence of dyslipidemia (94.44% *vs.* 73.08%).【Conclusions】Children and adolescent with craniopharyngioma exhibit a higher incidence of overweight/obesity than healthy peers preoperatively, which becomes more pronounced after surgery. This is accompanied by a high incidence of glucolipid metabolic disorders, placing these patients at increased risk of metabolic syndrome, which warrants clinical attention and regular metabolic assessment. In patients who develop postoperative obesity, particular attention should be given to the risk of glucose metabolism abnormalities, and further monitoring of glucose homeostasis is recommended.

Key words: children; adolescents; craniopharyngioma; overweight, obesity; glucolipid metabolism

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颅咽管瘤(craniopharyngioma, CP)是起源于垂体胚胎发生过程中残存扁平上皮细胞的先天性肿瘤,占儿童鞍区肿瘤的54%^[1]。随着诊疗技术的进步和多学科协作模式的应用,CP患者长期生存率显著提高,20年总生存率达80%^[2];儿童患者5年生存率达83%~96%,20年达62%^[3]。但由于肿瘤本身占位效应以及手术等治疗措施易损伤下丘脑-垂体,导致下丘脑-垂体功能障碍、代谢紊乱(如肥胖、糖脂异常)等,严重危害儿童及青少年患者的生长发育及生存质量^[4-8]。目前国内外关于CP儿童患者术后的管理主要集中于原发病的监测及垂体激素替代治疗,关于代谢的研究较少,主要为小样本单中心描述性报道,且随访时间较短,其影响因素、可能机制尚不明确。本研究回顾性分析了2006年1月至2024年2月在我院收治的0-18岁CP术后患者的平均随访7.45年的临床资料,系统探讨术后超

重/肥胖及糖脂代谢的临床特征与危险因素,旨在为早期识别和规范干预提供循证依据。

1 材料与方法

1.1 对象

2006年1月至2024年2月在中山大学孙逸仙纪念医院接受手术治疗且病理确诊为CP的0~18岁患者。纳入标准:①术后病理确诊为CP;②手术年龄在0~18岁。排除标准:①术后随访时间<6个月;②术前/术后身高、体质量、血糖、血脂、血压等随访数据严重缺失;③合并其他严重系统疾病或并发症的患者。最终纳入44例患者。本研究遵循我院医学伦理委员会制定的伦理学标准[批准文号:SYSKY-2024-463-01],并取得豁免知情同意。

1.2 资料收集

通过电子病历系统搜集患者围手术期和后期随访数据,包括:①人口学特征(性别、年龄等);②临床参数:术前症状、肿瘤影像特征、手术入路(经蝶/开颅等)等;③体格检查数据:身高、体质量、计算体质量指数(body mass index, BMI);④代谢指标:所有患儿均禁食一夜,次日清晨采集静脉外周血,检测指标包括空腹血糖(fasting blood glucose, FPG)、空腹胰岛素(fasting insulin, FINS)、计算胰岛素抵抗指数(HOMA-IR)、按临床特征及自愿原则行口服葡萄糖耐量试验(oral glucose tolerance test, OGTT)/胰岛素释放试验(口服葡萄糖粉 1.75 g/kg, 最大 75 g, 每克葡萄糖粉加水 3~4 mL, 在 5~10 min 服完, 分别在第 0 h、1 h、2 h 抽外周静脉血 4 mL 测定葡萄糖和胰岛素)、血脂谱[甘油三酯(triglyceride, TG)、总胆固醇(total cholesterol, TC)、低密度脂蛋白胆固醇(low density lipoprotein cholesterol, LDL-C)、高密度脂蛋白胆固醇(high density lipoprotein cholesterol, HDL-C)], 计算非高密度脂蛋白胆固醇(non-high-density lipoprotein cholesterol, nHDL)水平($nHDL=TC-HDL$)等;⑤内分泌评估:术前、术后垂体激素(growth hormone, GH/adrenocorticotrophic hormone, ACTH/thyroid stimulating hormone, TSH/luteinizing hormone, LH/follicle-stimulating hormone, FSH)水平及替代治疗方案。

1.3 评估标准

1.3.1 超重/肥胖 0~2岁,使用“身长的体质量”,根据世界卫生组织 2006 年的儿童生长发育标准, +2 SD~+3 SD 为超重, >+3 SD 为肥胖;2~5岁,参考“中国 0~18 岁儿童、青少年体块指数的生长曲线”中的 BMI 参考界值点, $p_{85} \leq BMI < p_{95}$ 为超重, $BMI \geq p_{95}$ 为肥胖;6~18岁参考 2018 年发布的《WS/T586-2018 学龄儿童青少年超重和肥胖筛查》的 BMI 参考界值点;≥18 岁时, BMI 均以 24 kg/m² 和 28 kg/m² 为超重、肥胖界值点。体质指数标准差积分(BMISDS)计算根据 BMI 标准差曲线图判断 [$BMISDS=(BMI-M)/sd$]^[9-11]。

1.3.2 糖代谢异常 参考标准为《儿童青少年 2 型糖尿病诊治专家共识》及美国 2024 ADA 糖尿病诊断标准^[12-13]。包括:(1)空腹血糖受损(impaired

fasting glucose, IFG): FPG 5.6~6.9 mmol/L; (2)糖耐量受损(impaired glucose tolerance, IGT): OGTT 2 h 血浆葡萄糖 7.8~11.0 mmol/L; (3)糖尿病(diabetes mellitus, DM): 满足其中一条即可诊断:①FPG≥7.0 mmol/L(空腹 8 h 后);②OGTT 2 h 血浆葡萄糖≥11.1 mmol/L;③典型高血糖临床症状+随机血浆葡萄糖≥11.1 mmol/L。(4)胰岛素抵抗(insulin resistance, IR):①FINS>15 mU/L;②OGTT 试验后胰岛素峰值>150 mU/L;③OGTT 试验后 120 min 时胰岛素>75 mU/L;④HOMA-IRI>2.5 {HOMA-IRI=[FINS(mU/L)×FPG(mmol/L)]/22.5}。

1.3.3 脂代谢异常 参考标准为《儿童脂质异常血症诊治专家共识》^[14]。包括①高 TC 血症: TC≥5.17 mmol/L;②高 TG 血症: 9 岁及以下儿童 TG≥1.12 mmol/L、10 岁及以上儿童 TG≥1.46 mmol/L;③混合型高脂血症: TC≥5.17 mmol/L 及 TG 升高;④高 nHDL 血症: TC-HDL≥3.74 mmol/L;⑤低 HDL 血症: HDL-C<1.03 mmol/L。

1.3.4 垂体激素评估标准及替代治疗方案 参考儿童及青少年颅咽管瘤术后内分泌管理指南^[6, 15-16]。

1.4 统计分析

采用 SPSS 25.0 进行统计分析,正态分布的计量资料采用($\bar{x} \pm s$)进行统计描述,组间比较用 t 检验;非正态资料采用 $M(P_{25}, P_{75})$ 进行统计描述,组间比较用秩和检验;计数资料或分类变量,采用例或百分数进行描述,采用卡方检验,或进行连续性校正或 Fisher 确切概率法进行组间变量比较;肥胖及糖脂代谢的影响因素采用 Logistic 回归分析;以 $P<0.05$ 为差异有统计学意义。由于样本量有限,本研究未进行多重检验校正,结果主要用于探索性分析。

2 结果

2.1 一般资料

44 例患者中男性 31 例(70.45%),女性 13 例(29.55%),手术时平均年龄为(8.57±4.25)岁,平均随访(7.45±4.64)年(范围 8 月~17.5 年),末次随访时平均年龄为(16.02±5.36)岁。43 例患儿获得术后 1 年内随访资料;25 例患儿随访时长达到或超过

队列平均随访时长7.45年。术前患儿临床表现以肿瘤占位相关症状为主, 颅高压32例(72.73%)、视力/视野受损21例(47.73%); 垂体内分泌功能障碍最常见为生长迟缓(10例, 22.73%)。术后颅高压

及肿瘤占位效应解除, 视力、视野恢复困难, 但日常生活不受影响。术后垂体内分泌功能障碍发生率显著升高, 主要表现为尿崩、甲状腺功能低下等(表1)。

表1 CP患儿手术前、后垂体内分泌功能障碍比较		
Table 1 Comparison of pituitary endocrine dysfunction in children with CP between before and after surgery		
[n/N(%), N=44]		
Index	Before surgery	After surgery
Normal pituitary function	29/44(65.91)	6/44(13.64)
GHD	10/44(22.73)	19/44(43.18)
Hypothyroidism	7/44(15.91)	24/44(54.55)
Adrenocortical insufficiency	5/44(11.36)	22/44(50.00)
Hypogonadism	4/44(9.09)	11/44(25.00)
Diabetes insipidus	3/44(6.82)	36/44(81.82)
Isolated pituitary hormone deficiency	6/44(13.64)	2/44(4.55)
Multiple pituitary hormone deficiency	9/44(20.45)	27/44(61.36)
Panhypopituitarism	0/44(0.00)	9/44(20.45)

2.2 患者超重/肥胖发生率分析

患者术前超重/肥胖有18例(40.91%), 其中超重12例(27.27%)、肥胖6例(13.64%)。术后第一年超重/肥胖发生率升高至68.18%, 其中超重8例(18.18%)、肥胖22例(50.00%; 图1)。术后患儿的BMI-SDS较术前升高(2.41 ± 3.06 vs. 0.38 ± 1.50 , $t=-5.37$, $P<0.01$)。

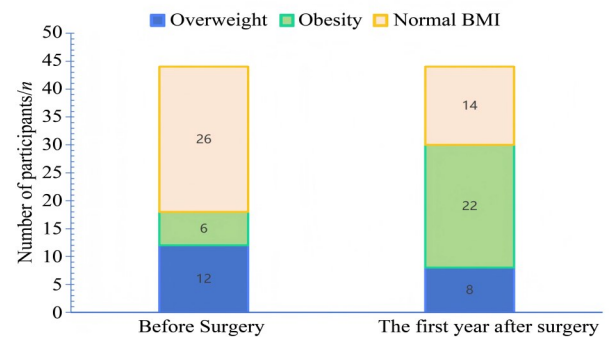


图1 手术前、后CP患者超重/肥胖人数变化(N=44)
Fig. 1 Changes in overweight/obesity in patients with CP before and after surgery (N=44)

2.3 患者术后糖代谢特征分析

在术后历次随访过程中, 糖稳态异常总体检出20/44例(45.45%), 主要为: 44例患者有FPG资料,

有2例(4.55%)患者存在IFG; 有20例患者检测了FINS, 11例(55.00%)存在IR; 有12例患者进一步接受了OGTT/胰岛素释放试验检查, 其中2例(16.67%)存在IGT, 3例(25.00%)患者达到DM诊断标准, 有12/12例(100%)存在胰岛素释放异常, 5/12例(41.67%)存在高胰岛素血症。糖代谢指标水平如表2。

2.4 患者术后脂代谢特征分析

在术后历次随访过程中, 脂代谢异常检出36/44例(81.82%), 主要为: 高TC血脂22例(50.00%)、高TG血症29例(65.91%)、低HDL-C血症22例(50.00%)、高nHDL血症29例(65.91%)、混合型高脂血症13例(29.55%)。脂代谢指标水平如表3。

2.5 CP患者术后超重/肥胖的影响因素分析

以术后超重/肥胖为因变量, 性别、年龄、肿瘤部位、手术入路、垂体激素缺乏等为自变量行进行logistic分析, 发现术前超重/肥胖、术后糖代谢异常是术后超重/肥胖的影响因素(表4)。

2.6 术后超重/肥胖与BMI正常患者的糖脂代谢特征对比分析

术后超重/肥胖患者与BMI正常患者的性别组成、手术年龄、肿瘤部位及手术方式等差异无统计

表2 CP患儿术后糖代谢特征

Table 2 Characteristics of glucose metabolism in pediatric patients with CP after surgery

[N=44,M(P_{25} , P_{75})]

Index	Abnormal glucose homeostasis group(n=20)	Normal glucose homeostasis group(n=24)
FPG/(mmol/L)	4.6(4.08,4.78)	4.2(3.55,4.85)
FINS/(mU/L)	10.25(5.08,23.41)	2.69(1.96,8.36)
HOMA-IR	2.07(0.95,4.49)	0.39(0.3,1.67)

FPG: fasting blood glucose(reference range, 3.90 to 5.60 mmol/L); FINS: fasting insulin(reference range, 3.00 to 25.00 mU/L); HOMA-IR: homeostatic model assessment of insulin resistance(reference range, <2.5).

表3 CP患儿术后脂代谢特征

Table 3 Characteristics of lipid metabolism in pediatric patients with CP after surgery

[mmol/L,M(P_{25} , P_{75})]

Index	Abnormal lipid metabolism group(n=36)	Normal lipid metabolism group(n=8)
TC	5.13(4.46,5.93)	3.77(3.68,4.45)
TG	1.58(1.20,2.97)	1.14(0.97,1.32)
HDL-C	1.07(0.8,1.38)	0.98(0.90,1.82)
LDL-C	3.17(2.71,3.92)	2.11(1.90,2.71)
n-HDL-C	3.95(3.37,4.85)	2.63(2.41,3.08)

TC: total cholesterol (reference range, 2.90 to 6.00 mmol/L); TG: triglycerides(reference range, 0.56 to 2.30 mmol/L); HDL-C: high-density lipoprotein cholesterol(reference range, 1.03 to 2.07 mmol/L); LDL: low-density lipoprotein cholesterol(reference range, <3.4 mmol/L); n-HDL-C: non-high-density lipoprotein cholesterol(reference range, <3.74 mmol/L).

表4 CP患者术后超重/肥胖的影响因素

Table 4 Factors associated with overweight/obesity in patients with CP after surgery

Index	b	S_b	Wald	P	OR	OR 95% CI
Gender	-0.171	0.665	0.066	0.797	0.843	0.229-3.101
Age	0.12	0.078	2.325	0.127	1.127	0.966-1.315
Tumor location	0.052	0.402	0.017	0.897	1.053	0.479-2.318
Surgical approach	20.877	2 3205.4	0	0.999	1.17E+09	0-N.A.
Overweight/obesity before surgery	1.751	0.811	4.661	0.031*	5.762	1.175-28.251
Hypothyroidism after surgery	1.232	1.163	1.122	0.29	3.429	0.351-33.518
Adrenocortical insufficiency after surgery	1.504	1.143	1.733	0.188	4.5	0.479-42.248
GHD after surgery	21.056	2 3205.43	0	0.999	1.4E+09	0-N.A.
Abnormal glucose homeostasis after surgery	1.977	0.717	7.609	0.006*	7.219	1.772-29.406

学意义;但在垂体各轴激素替代平稳时,超重/肥胖患者的FINS水平、HOMA-IR、糖代谢异常发生率高于BMI正常的患者;而血脂谱及脂代谢异常发生率差异无统计学意义(表5)。

3 讨论

3.1 儿童及青少年CP患者手术前后临床特征

CP是一种先天性颅内良性肿瘤,通常生长于

表5 CP术后超重/肥胖与BMI正常患者糖脂代谢特征的比较			
Table 5 Comparison of glucolipid metabolism characteristics in patients with CP after surgery between overweight/obesity and normal BMI	[n, M(P ₂₅ , P ₇₅), n/N(%)]		
	Overweight/obesity	Normal BMI	P
Gender/[N(F/M)]	18(6/12)	26(7/19)	0.65
Age at surgery/years	7.25±3.35	9.48±4.61	0.09
Tumor location (sellar/suprasellar/sellar+suprasellar/other)	5/4/3/6	9/4/5/8	0.77
Surgical approach(microsurgery/craniotomy)	6/12	7/19	0.18
FPG/(mmol/L)	4.6(4.3,4.8)	4.1(3.53,4.85)	0.10
FINS/(mU/L)	14.22(5.77,31.17)	3.57(2.40,10.41)	0.01
HOMA-IR	2.88(1.21,4.91)	0.52(0.35,2.1)	0.01
Abnormal glucose Homeostasis	14/18(77.78)	6/26(23.08)	0.00
TC/(mmol/L)	4.54(3.94,5.26)	5.2(4.37,6.16)	0.12
TG/(mmol/L)	1.55(1.31,2.37)	1.39(1.04,3.18)	0.81
HDL-C/(mmol/L)	1.02(0.76,1.14)	1.16(0.9,1.61)	0.09
LDL-C/(mmol/L)	2.81(2.49,3.48)	3.08(2.55,4.1)	0.32
n-HDL/(mmol/L)	3.61(2.87,4.23)	3.88(3.04,4.93)	0.35
Dyslipidemia	17/18(94.44)	19/26(73.08)	0.38

鞍区,与下丘脑、视交叉、垂体柄位置关系密切,紧邻重要神经血管结构,其特殊的侵袭性生长方式对邻近组织造成不可逆破坏,引起头痛、呕吐、视野受损、视力下降等颅内占位相关表现,以及生长迟缓、尿崩等垂体内分泌障碍表现。本研究发现,44例患者的平均手术年龄为(8.57±4.25)岁,与文献报道的儿童高发年龄为5~15岁相符;患者术前就诊的主要原因因为头痛、视力障碍等颅内占位表现,而以垂体功能障碍就诊的比例偏低,其原因可能与多饮/多尿、生长迟缓、体质量增加快等起病隐匿,不易引起家长及医生重视有关。手术是CP的一线治疗手段,能快速缓解颅高压等占位效应,但侵入性操作进一步破坏了垂体及邻近组织的结构和功能,导致下丘脑-垂体功能障碍加剧;因此儿童及青少年CP患者术后临床表现多以垂体内分泌功能障碍为突出表现^[17]。

3.2 CP患者超重/肥胖的高发性及机制

肥胖是CP术后最常见并发症之一,肥胖的严重程度和下丘脑损伤程度相关^[18],有研究报道20%的患者在术前即出现肥胖,而术后高达65%~80%^[19]。本研究发现,CP患儿术前超重/肥胖的发

生率高达40.91%,显著高于正常儿童^[20],其中以超重尤为突出(27.27%),提示术前部分患儿可能已存在下丘脑受累或垂体轴功能受损相关的代谢异常。术后第一年超重/肥胖发生率急剧升高至68.18%,肥胖率显著高于超重(50.00% vs. 18.18%),且患者术后BMI-SDS较术前升高,提示CP相关的代谢异常可能受疾病本身及手术治疗的双重影响。

机制方面,首先颅咽管瘤的生长方式导致垂体组织受压、破坏^[21-22],垂体功能障碍导致GH/ACTH/TSH轴异常/紊乱,引发基础代谢率降低及脂肪代谢障碍,这一机制可解释术前以超重为主的代谢表型^[23]。其次,手术相关的下丘脑损伤,尤其是腹内侧核-弓状核回路破坏,会导致瘦素抵抗和神经肽Y过度表达,诱发暴食行为和交感神经抑制(静息能耗降低),从而形成下丘脑性肥胖的病理过程^[24]。既往研究表明,不同的治疗方式或术中对垂体的处理方式不同,对术后垂体功能减退的发生率可能会产生影响^[6]。因此,外科医生在切除肿瘤的前提下,尽可能地保护垂体/垂体柄以及下丘脑的正常结构,可能对患者术后的代谢有积极作用。

3.3 CP患者糖代谢异常的高发与下丘脑损伤相关

本组患儿术后糖代谢异常总体发生率为45.45%,以胰岛素抵抗、高胰岛素血症、胰岛素释放异常为主要特征;且与体质量正常的CP术后患者相比,超重/肥胖患者的空腹胰岛素水平(14.22 mU/L vs. 3.57 mU/L)、HOMA-IR(2.88 vs. 0.52)及异常糖代谢发生率(77.78% vs. 23.08%)更高,提示下丘脑肥胖的患者存在糖代谢紊乱的高发风险。机制除了肥胖所致胰岛素抵抗外,可能还包括:①CP相关的垂体激素缺乏导致基础代谢率下降、糖利用降低及脂肪动员受阻,进而引起或加重胰岛素抵抗^[25-27];②下丘脑腹内侧核损伤,对迷走神经的抑制减弱,导致迷走神经过度活跃,刺激胰岛 β 细胞过度分泌胰岛素,形成高胰岛素血症^[24];③医源性激素替代不恰当,尤其是过量的糖皮质激素替代治疗,也可能进一步加重胰岛素抵抗。

3.4 脂代谢紊乱广泛存在,但不完全与肥胖有关

既往研究提示,成年CP患者术后脂代谢异常的发生率高达76.9%,主要表现为TC、TG及LDL-C水平升高^[8]。但儿童CP术后脂代谢的研究仍有限,罗杰等^[27]对20例CP术后儿童患者的研究显示,血脂代谢异常的患儿高达85%,包括高TC血症(50%),高TG血症(65%),混合型高脂血症(40%),低HDL-C血症(40%),高LDL-C血症(50%)。Du等^[28]在对60名CP患儿的回顾性分析亦证实,与健康儿童相比,CP患儿的TC、TG、LDL明显增高,HDL水平明显下降,脂质代谢异常的发病率更高。本研究中,CP患儿脂代谢异常的发生率为81.82%,主要为高n-HDL血脂(65.91%)、高TG血症

(65.91%)、低HDL血症(50.00%)、混合型高脂血症13例(29.55%),与既往研究结果相符。

有观点认为脂代谢紊乱主要继发于下丘脑性肥胖。但本研究进一步发现,即使在BMI正常的CP患儿中,脂代谢异常的发生率亦高达73.08%(19/26例),与超重/肥胖组(94.44%,17/18例)差异无统计学意义($P>0.05$),提示该类患儿的脂质代谢异常并非完全与肥胖相关。除下丘脑损伤外,垂体功能障碍(如皮质醇、甲状腺素缺乏)亦可能通过降低基础代谢、减少脂肪动员和糖利用等途径参与脂代谢紊乱。本研究发现,即使术后糖皮质激素、甲状腺素替代平稳,血脂异常的发生率仍高达80%以上,提示可能存在其他机制。

3.5 局限性

本研究存在一些局限性。首先,本研究属于单中心回顾性研究,存在回顾性偏倚。并且因为回顾性收集病例数据,存在部分病例数据缺失,未能进一步描述CP患儿术前糖脂代谢的特点、下丘脑综合征的临床特征、术后激素替代详细方案并作进一步分析。其次,样本量小,未能进一步行亚组对比分析,如不同年龄段及青春发育分期的代谢特征对比。最后,本研究为描述性分析,未做机制探讨,尤其关于CP患儿术后脂代谢异常的原因及影响因素未进行深入讨论。这均提示后续需扩大样本量,规范检验项目及随访指标,进一步分析CP患儿术后糖脂代谢特点及研究相关影响因素。

综上,本研究显示,儿童及青少年CP术后存在较高比例的超重/肥胖及糖脂代谢异常,其中肥胖患儿糖代谢异常更为突出。应将体质量管理与糖脂代谢监测纳入长期随访策略。

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