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病毒性肝炎肝硬化患者早期肝细胞癌代谢变化 及其与衰老和酶活性的关系

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摘要 **目的** 探讨病毒性肝炎肝硬化患者早期肝细胞癌代谢变化及其与衰老和酶活性的关系。**方法** 以140例患者为研究对象,根据诊断结果分为肝硬化组(LC组)、肝硬化肝细胞癌组(CLH组)、早期肝细胞癌组(HCC组)和对照组,每组35例。采用氢质子磁共振波谱($^1\text{H-MRS}$)测量肝细胞代谢物水平,采用广义估计方程分析代谢物水平变化的潜在风险因素。**结果** LC组血清碱性磷酸酶(ALP)水平、CLH组血清天门冬氨酸氨基转移酶(AST)、乳酸脱氢酶(LDH)、ALP、葡萄糖水平较对照组升高($P < 0.05$),CLH组血清AST、葡萄糖水平较LC组升高($P < 0.05$)。LC组、CLH组乳酸+三酰甘油(Lac+TG)(1.3 ppm)水平较对照组、HCC组降低($P < 0.05$)。CLH组胆碱(Cho)(3.2 ppm)水平较对照组升高($P < 0.05$),HCC组Cho(3.2 ppm)水平较LC、CLH、对照组升高($P < 0.05$)。LC、CLH和HCC组三酰甘油(TG)(0.9 ppm)及TG(2.1 ppm)水平较对照组降低($P < 0.05$)。

LC、CLH和HCC组患者中,Lac+TG(1.3 ppm)水平与年龄、LDH呈正相关,Cho(3.2 ppm)水平与ALP呈正相关。Lac+TG及Cho代谢变化的相关危险因素是年龄60~80岁。交互作用分析结果示,Lac+TG水平与40~50岁男性或女性的HCC及60~80岁男性或女性的LC、CLH、HCC有关($P < 0.05$)。Cho水平与60~80岁男性或女性的HCC有关($P < 0.05$)。**结论** 高Lac+TG和Cho水平可能与病毒性肝炎肝硬化患者早期肝细胞癌代谢变化有关。此外,肝细胞癌中Lac+TG水平与较大年龄和LDH呈正相关。

关键词 病毒性肝炎;肝硬化;肝细胞癌;氢质子磁共振波谱
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肝细胞癌(hepatocellular carcinoma,HCC)是最常见的原发性肝恶性肿瘤,常由肝炎和肝硬化(liver cirrhosis,LC)发展而来^[1]。LC患者的肝细胞癌变在很大程度上是一个从再生结节到HCC的多步演变过程,但早期诊断HCC仍具有挑战性^[2]。肝活检是诊断慢性肝病金标准,但因其具有侵入性,使用常受到限制^[3]。氢质子磁共振波谱(proton magnetic resonance spectroscopy, $^1\text{H-MRS}$)是一种非侵入性成

that required evisceration or enucleation (24 patients) with those that received salvaging therapies (97 patients) were compared. Age, sex, medication history, past medical history, clinical manifestation, Leukocyte counts and treatment progression were retrospectively analyzed. **Results** Twenty four eyes(19.8%) underwent enucleation or evisceration. The proportion of corneal ulcerative endophthalmitis (33.3%) and endogenous endophthalmitis (25.0%) in evisceration or enucleation group was greater than that in the salvaging group (1.0%, 4.1%) ($P < 0.001$). The group of eviscerated or enucleated eyes was older ($P < 0.05$), had poorer initial visual acuity [(2.9 ± 0.2) LogMAR vs (2.3 ± 0.5) LogMAR, $P < 0.001$], had longer duration before intervention (15.8 d vs 4.6 d, $P < 0.05$), and had more Leukocyte counts [$(12.8 \pm 5.6) \times 10^9/\text{L}$ vs $(9.1 \pm 3.3) \times 10^9/\text{L}$, $P < 0.005$]. With Logistic regression analysis, corneal ulcer, endogenous endophthalmitis, initial vision, leukocyte counts, duration before intervention were the risk factor for evisceration or enucleation ($OR = 343.283$, $OR = 22.608$, $OR = 1920.384$, $OR = 1.341$, $OR = 1.167$). **Conclusion** The most common cause of evisceration or enucleation caused by infectious endophthalmitis are infections from corneal ulcer and from endogenous source. The risk factors for endophthalmitis requiring evisceration or enucleation would be considered to be corneal ulcer endophthalmitis, endogenous endophthalmitis, initial vision, leukocyte counts, and duration before intervention.

Key words infectious endophthalmitis; evisceration; enucleation; risk factors

像技术^[4]。目前,¹H-MRS 已广泛应用于中枢神经系统、乳腺和前列腺,但在肝脏中的应用却相对不足^[5]。LC、肝硬化肝细胞癌(cirrhotic liver with hepatocellular carcinoma, CLH)和 HCC 患者的衰老和酶活性可能会影响细胞代谢物的形成机制。因此,该研究旨在探讨病毒性肝炎肝硬化患者早期肝细胞癌代谢变化及其与衰老和酶活性的关系。

1 材料与方法

1.1 病例资料 选取 2018 年 6 月—2020 年 6 月在陆军军医大学第一附属医院感染病科就诊的 140 例患者为研究对象。根据诊断结果分为 4 组,其中 LC 组 35 例,CLH 组 35 例,HCC 组 35 例,同期就诊的健康体检者(对照组)35 例。排除标准:① HCC 最长直径 < 2 cm;② 肝脏中脂肪沉积;③ 酒精性肝硬化。本研究经陆军军医大学第一附属医院伦理委员会批准,所有患者均知情同意并签署知情同意书。

1.2 ¹H-MRS 检查 采用 3.0 T 超导磁共振仪(Siemens 公司,德国)。使用 3D T1 高分辨力各向同性容积激发脉冲序列获取 T1 加权图像。采用激励回波采集模式序列进行高速 T2 校正多回波¹H-MRS 测量,用于定量肝脂肪含量。

具有长 T2 弛豫时间的乳酸(lactic acid, Lac)和胆碱(choline, Cho)代谢物,采用点分辨波谱序列进行单体素¹H-MRS 测量。在感兴趣容积的上下、左右、前后界添加 6 个饱和带,以消除周围组织造成的影响。检查前先进行自动预扫描完成匀场及抑水,预扫描完成后以 T2WI 图像用作¹H-MRS 定位。对照组和 LC 组中将体素置于在肝右叶上,CLH 及 HCC 组中将一个体素置于肿瘤实质内,避开肿瘤坏死区域,将另一个体素置于肝实质内,放置远离肝脏

边缘,并避开大血管及肝内胆管。

扫描完成后,将波谱数据传输至西门子工作站进行调节和校正,记录主要的肝细胞代谢物 Lac + TG 峰(1.3 ppm)、Cho 峰(3.2 ppm)及 TG 峰(0.9 ppm 和 2.1 ppm)的峰下面积和各相关峰比值,残留水峰在 4.7 ppm 处用作内部参考。通过使用线性最小二乘拟合算法校正 T2 的效应,得到校正后水峰下面积($S_{\text{水}}$)和脂肪峰下面积($S_{\text{脂}}$),计算肝脏脂肪分数(hepatic fat fraction, HFF)。HFF = $S_{\text{脂}} / (S_{\text{脂}} + S_{\text{水}}) \times 100\%$ ^[6]。

1.3 观察指标 主要观察指标包括:①人口统计学指标:年龄、性别;②生化指标:天门冬氨酸氨基转移酶(aspartate aminotransferase, AST)、丙氨酸氨基转移酶(alanine aminotransferase, ALT)、乳酸脱氢酶(lactate dehydrogenase, LDH)、碱性磷酸酶(alkaline phosphatase, ALP)、葡萄糖、白蛋白、总胆红素;③代谢指标:三酰甘油(triglycerides, TG)、HFF、Lac、Cho。

1.4 统计学处理 采用 SPSS 19.0 软件进行数据分析。计量资料用 $\bar{x} \pm s$ 表示,多组间比较用单因素方差分析,进一步两两比较采用 LSD 法。计数资料用 $n(\%)$ 表示,多组间比较用 χ^2 检验。相关分析采用 Pearson 相关性分析。代谢物水平变化的潜在风险因素采用广义估计方程分析。检验水准 $\alpha = 0.05$ 。

2 结果

2.1 各组间人口统计学及血清生化指标比较 LC 组血清 ALP 水平较对照组升高,CLH 组血清 AST、LDH、ALP、葡萄糖水平较对照组升高,CLH 组血清 AST、葡萄糖水平较 LC 组升高,差异有统计学意义($P < 0.05$)。见表 1。

表 1 不同组间人口统计学及血清生化指标比较($\bar{x} \pm s$)

临床参数	对照组($n=35$)	LC 组($n=35$)	CLH 组($n=35$)	F 值	P 值
年龄(岁)	58.14 ± 13.27	55.25 ± 13.92	62.07 ± 14.16	1.727	0.184
性别(男/女)	18/17	17/18	17/18	0.076	0.963
AST(IU/L)	38.42 ± 22.57	47.58 ± 23.19	66.64 ± 38.89 ^{*#}	6.804	0.002
ALT(IU/L)	34.28 ± 18.83	36.20 ± 18.53	45.21 ± 27.09	1.997	0.142
LDH(IU/L)	387.45 ± 56.43	421.60 ± 117.69	455.00 ± 104.68 [*]	3.424	0.037
ALP(IU/L)	93.59 ± 28.93	119.15 ± 30.75 [*]	128.92 ± 68.84 [*]	4.289	0.017
葡萄糖(mg/dl)	101.06 ± 15.71	107.88 ± 16.86	135.98 ± 22.94 ^{*#}	27.216	<0.001
TG(mg/dl)	141.95 ± 45.41	128.57 ± 45.13	122.67 ± 46.65	1.306	0.277
白蛋白(g/dl)	4.03 ± 1.48	4.22 ± 1.59	3.88 ± 1.36	0.375	0.688
总胆红素(mg/dl)	0.99 ± 0.50	1.19 ± 0.45	1.39 ± 0.79	2.957	0.058

与对照组比较:^{*} $P < 0.05$;与 LC 组比较:[#] $P < 0.05$

2.2 各组间脂肪变性程度及肝细胞代谢物水平比较 LC组、CLH组 Lac + TG (1.3 ppm) 水平较对照组、HCC组降低, 差异有统计学意义 ($P < 0.05$)。CLH组 Cho (3.2 ppm) 水平较对照组升高, HCC组 Cho (3.2 ppm) 水平较对照组、LC组、CLH组升高, 差异有统计学意义 ($P < 0.05$)。LC组、CLH组、HCC组 TG (0.9 ppm) 及 TG (2.1 ppm) 水平较对照组降低, 差异有统计学意义 ($P < 0.05$)。见表2。

2.3 患者肝细胞代谢物水平与年龄及酶活性的相关性 LC、CLH和HCC组患者中, Lac + TG (1.3 ppm) 水平与年龄、LDH呈正相关, Cho (3.2 ppm) 水平与ALP呈正相关。见表3。

2.4 Lac + TG及Cho代谢变化相关危险因素的广义估计方程分析 Lac + TG及Cho代谢变化的相关危险因素是年龄60~80岁。交互作用分析结果示, Lac + TG水平与40~50岁男性或女性的HCC及60~80岁男性或女性的LC、CLH、HCC有关 ($P < 0.05$)。Cho水平与60~80岁男性或女性的HCC有关 ($P < 0.05$)。见表4。

3 讨论

与LC组比较, HCC组 Lac + TG水平升高。此

外, 与对照组比较, LC、CLH和HCC组 TG (0.9 ppm) 及 TG (2.1 ppm) 水平降低。尽管在1.3 ppm的Lac信号与TG重叠, 但Lac水平仍有助于反映出CLH和HCC之间代谢模式的差异。Kim et al^[7]发现非酒精性脂肪肝患者也出现特定Lac + TG峰。研究表明^[8], Lac在HCC癌变和转移过程中在癌细胞中积累, 使肿瘤的细胞外pH始终保持酸性。酸性厌氧代谢会导致内环境紊乱, 这可能增加导致肿瘤发生的遗传突变率^[9]。根据几项超极化¹³C MRS研究^[10-11], 肝损伤和HCC中Lac升高, 并且HCC中LDH-A水平高于正常肝脏, 表明HCC中Lac水平升高可能归因于LDH-A酶水平增加。本研究发现, LC、CLH和HCC组患者中Lac + TG (1.3 ppm) 水平与LDH呈正相关, 提示LDH水平升高可能与Lac代谢异常有关。这表明Lac + TG水平可能是一种反映LC背景下HCC发生过程中特异性代谢的潜在生物标志物。

本研究显示, HCC组 Cho (3.2 ppm) 水平高于LC、CLH、对照组。Cho是与磷脂代谢相关的细胞膜中的重要成分, 因此是细胞增殖的有效指标^[12]。Cho增加可能会通过激活编码与胆碱代谢相关酶的基因来促进HCC细胞的增殖与侵袭^[13]。因此,

表2 各组间脂肪变性程度及肝细胞代谢物水平比较 ($\bar{x} \pm s$)

组别	HFF (%)	Lac + TG (1.3 ppm)	Cho (3.2 ppm)	TG (0.9 ppm)	TG (2.1 ppm)
对照	4.21 ± 2.07	3.12 ± 1.10	0.05 ± 0.02	0.43 ± 0.18	0.37 ± 0.11
LC	4.58 ± 2.06	0.48 ± 0.12 *	0.08 ± 0.04	0.13 ± 0.06 *	0.07 ± 0.02 *
CLH	4.51 ± 2.09	0.50 ± 0.12 *	0.11 ± 0.05 *	0.13 ± 0.06 *	0.05 ± 0.02 *
HCC	3.51 ± 1.48	2.72 ± 1.04 [#]	0.24 ± 0.11 ^{*#}	0.12 ± 0.05 *	0.05 ± 0.02 *
F值	1.780	96.107	42.098	62.731	192.238
P值	0.155	<0.001	<0.001	<0.001	<0.001

与对照组比较: * $P < 0.05$; 与LC组比较: [#] $P < 0.05$; 与CLH组比较: [&] $P < 0.05$

表3 LC、CLH和HCC组患者肝细胞代谢物水平与年龄及酶活性的相关性

代谢物	年龄	AST	ALT	LDH	ALP	葡萄糖	TG	白蛋白	总胆红素
Lac + TG (1.3 ppm)									
r值	0.315	0.031	0.076	0.298	-0.084	0.115	-0.145	0.047	-0.076
P值	0.002	0.780	0.480	0.004	0.433	0.290	0.440	0.674	0.483
Cho (3.2 ppm)									
r值	0.113	0.125	0.116	0.049	0.340	-0.124	-0.156	0.097	-0.155
P值	0.312	0.232	0.180	0.652	0.002	0.248	0.403	0.364	0.163
TG (0.9 ppm)									
r值	0.012	-0.116	-0.028	0.105	0.156	0.040	-0.052	0.116	-0.094
P值	0.689	0.372	0.831	0.412	0.212	0.741	0.816	0.374	0.466
TG (2.1 ppm)									
r值	0.024	0.024	0.177	-0.051	-0.082	-0.005	-0.091	0.018	-0.133
P值	0.531	0.850	0.165	0.682	0.504	0.968	0.682	0.901	0.304

表4 Lac + TG 及 Cho 代谢变化相关危险因素的广义估计方程分析

变量	Lac + TG 水平			Cho 水平		
	OR	95% CI	P 值	OR	95% CI	P 值
40 ~ 50 岁	1.000			1.000		
60 ~ 80 岁	1.506	1.104 ~ 2.056	0.008	1.048	1.005 ~ 1.092	0.022
女性 × 40 ~ 50 岁 × LC	1.000			1.000		
女性 × 40 ~ 50 岁 × CLH	1.035	0.955 ~ 1.120	0.402	1.005	0.970 ~ 1.038	0.821
女性 × 40 ~ 50 岁 × HCC	4.760	3.072 ~ 7.390	0.001	1.064	0.991 ~ 1.130	0.070
女性 × 60 ~ 80 岁 × LC	1.117	1.045 ~ 1.190	0.001	0.942	0.905 ~ 0.993	0.066
女性 × 60 ~ 80 岁 × CLH	1.134	1.005 ~ 1.284	0.043	1.026	0.993 ~ 1.058	0.120
女性 × 60 ~ 80 岁 × HCC	5.270	3.262 ~ 8.534	0.001	1.253	1.160 ~ 1.358	0.001
男性 × 40 ~ 50 岁 × LC	1.015	0.933 ~ 1.094	0.750	0.991	0.953 ~ 1.023	0.611
男性 × 40 ~ 50 岁 × CLH	1.045	0.953 ~ 1.150	0.321	1.033	0.980 ~ 1.088	0.174
男性 × 40 ~ 50 岁 × HCC	7.422	4.480 ~ 12.419	0.001	1.185	1.028 ~ 1.374	0.056
男性 × 60 ~ 80 岁 × LC	1.136	1.022 ~ 1.264	0.012	1.039	0.971 ~ 1.113	0.237
男性 × 60 ~ 80 岁 × CLH	1.094	1.001 ~ 1.224	0.047	1.000	0.973 ~ 1.038	0.982
男性 × 60 ~ 80 岁 × HCC	15.201	7.765 ~ 29.861	0.001	1.155	1.057 ~ 1.267	0.001

HCC 中高水平的 Cho 可能是持续细胞增殖的指标^[14]。LC、CLH 和 HCC 组患者中 Cho(3.2 ppm)水平与 ALP 呈正相关。表明 Cho 可能是与 HCC 相关细胞增殖的潜在生物标志物。此外,本研究广义估计方程分析结果示 Lac + TG 及 Cho 代谢变化的相关危险因素是年龄,尤其是 60 ~ 80 岁。作为 Lac + TG 水平改变的危险因素,HCC 患者中年龄超过 60 岁的男性 OR 值是 40 ~ 50 岁男性 OR 值的 2 倍,Cho 水平也与 60 ~ 80 岁男性或女性的 HCC 有关。提示年龄超过 60 岁的肝硬化患者,随着 Lac + TG 和 Cho 水平的改变,可能发生 HCC 的风险更高。

综上所述,体内¹H-MRS 可用于量化肝 Lac + TG 和 Cho 的水平,其中高 Lac + TG 和 Cho 水平可能与病毒性肝炎肝硬化患者早期肝细胞癌代谢变化有关。此外,肝细胞癌中 Lac + TG 水平与较大年龄和 LDH 呈正相关。

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Metabolic changes of early hepatocellular carcinoma in patients with liver cirrhosis caused by viral hepatitis and its relationship with aging and enzyme activity

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Abstract *Objective* To investigate the metabolic changes of early hepatocellular carcinoma in patients with cirrhosis caused by viral hepatitis and its relationship with aging and enzyme activity. *Methods* According to the diagnosis results, a total of 140 patients were divided into liver cirrhosis group (LC group), hepatocellular carcinoma with liver cirrhosis group (CLH group), early hepatocellular carcinoma group (HCC group) and control group, with 35 cases in each group. The proton magnetic resonance spectroscopy ($^1\text{H-MRS}$) was used to measure liver cell metabolite levels, and the generalized estimating equation was used to analyze potential risk factors for changes in metabolite levels. *Results* Serum alkaline phosphatase (ALP) levels in the LC group, serum aspartate aminotransferase (AST), lactate dehydrogenase (LDH), ALP, and glucose levels in the CLH group were higher than those in the control group ($P < 0.05$), and the serum AST and glucose levels in the CLH group were higher than those in the LC group ($P < 0.05$). The levels of lactic acid and triglycerides (Lac + TG) (1.3 ppm) in LC group and CLH group were lower than those in control group and HCC group ($P < 0.05$). The level of choline (Cho) (3.2 ppm) in the CLH group was higher than that in the control group ($P < 0.05$), and the level of Cho (3.2 ppm) in the HCC group was higher than that in the control, LC and CLH groups ($P < 0.05$). The levels of triglycerides (TG) (0.9 ppm) and TG (2.1 ppm) in the LC, CLH and HCC groups were lower than those in the control group ($P < 0.05$). In the LC, CLH and HCC groups, the Lac + TG (1.3 ppm) level was positively correlated with age, LDH, and the Cho (3.2 ppm) level was positively correlated with ALP ($r = 0.340$, $P = 0.002$). The related risk factors for changes in Lac + TG and Cho metabolism were age 60–80 years. The results of interaction analysis showed that Lac + TG levels were related to HCC in men or women aged 40 to 50, and LC, CLH, HCC in men or women aged 60 to 80 ($P < 0.05$). The Cho level was related to HCC in men or women aged 60 to 80 ($P < 0.05$). *Conclusion* The high levels of Lac + TG and Cho may be related to the metabolic changes of early hepatocellular carcinoma in patients with cirrhosis caused by viral hepatitis. In addition, the level of Lac + TG in hepatocellular carcinoma is positively correlated with older age and LDH.

Key words viral hepatitis; liver cirrhosis; hepatocellular carcinoma; $^1\text{H-MRS}$