

酒精性肝病诊疗指南 (2010 年修订版)

中华医学会肝病学会脂肪肝和酒精性肝病学组

【关键词】 肝疾病, 酒精性; 诊断; 治疗; 指南[文献类型]

Guidelines for management of alcoholic liver disease: an updated and revised edition *The Chinese National Workshop on Fatty Liver and Alcoholic Liver Disease for the Chinese Liver Disease Association.*

【Key words】 Liver disease, alcoholic; Diagnosis; Therapy; Guidebooks[Publication type]

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酒精性肝病是由于长期大量饮酒导致的肝脏疾病。初期通常表现为脂肪肝, 进而可发展成酒精性肝炎、肝纤维化和肝硬化。严重酗酒时可诱发广泛肝细胞坏死, 甚至肝功能衰竭。酒精性肝病是我国常见的肝脏疾病之一, 严重危害人民健康。为进一步规范酒精性肝病的诊断与治疗, 中华医学会肝病学会脂肪肝和酒精性肝病学组组织国内有关专家, 在参考国内外最新研究成果和相关诊疗共识的基础上, 对 2006 年制订的《酒精性肝病诊疗指南》进行修改和补充。本次修订主要按照循证医学的原则, 其中推荐意见所依据的证据等级共分为 3 个级别 5 个等次^[1], 文中以括号内斜体罗马数字表示。

本《指南》旨在帮助临床医师对酒精性肝病诊断与治疗作出正确决策, 并非强制性标准, 临床医师在针对某一具体患者时, 应充分了解本病的最佳临床证据和现有医疗资源, 并在全面考虑患者的具体病情及其意愿的基础上, 根据自己的知识和经验, 制定合理的诊疗方案。

一、流行病学

我国尚缺乏酒精性肝病的全国性大规模流行病学调查资料, 但地区性流行病学调查显示我国饮酒人群和酒精性肝病的患病率有上升趋势。华北地区流行病学调查显示, 从 20 世纪 80 年代初到 90 年代初, 嗜酒者在一般人群中的比例从 0.21% 升至 14.3%^[2]; 本世纪初, 南方及中西部省份流行病学调查显示饮酒人群增至 30.9%~43.4%^[2-3](Ⅲ)。

饮酒人群中一部分嗜酒者或饮酒过量的人群出现酒精相关健康问题, 其中酒精性肝病是酒精所致的最常见的脏器损害。本世纪初, 南方及中西部省份酒精性肝病流行病学调查

资料显示, 成人群体酒精性肝病患病率为 4.3%~6.5%^[3-5](Ⅲ)。酒精性肝病占同期肝病住院患者的比例在不断上升, 从 1991 年的 4.2% 增至 1996 年的 21.3%; 酒精性肝硬化在肝硬化的病因构成比从 1999 年的 10.8% 上升到 2003 年的 24.0%^[6-7](Ⅲ)。酒精所致的肝脏损害已经在中国成为一个不可忽视的问题。

二、危险因素

影响酒精性肝损伤进展或加重的因素较多, 目前国内外研究已经发现的危险因素主要包括: 饮酒量、饮酒年限、酒精饮料品种、饮酒方式、性别、种族、肥胖、肝炎病毒感染、遗传因素、营养状况等。

根据流行病学调查资料, 酒精所造成的肝损伤是有阈值效应的, 即达到一定饮酒量或饮酒年限, 就会大大增加肝损害风险^[8](Ⅲ)。然而, 由于个体差异较大, 也有研究显示饮酒与肝损害的剂量效应关系并不十分明确^[8-10](Ⅲ)。

酒精饮料品种较多, 不同的酒精饮料对肝脏所造成的损害也有差异^[11-12](Ⅲ)。饮酒方式也是酒精性肝损伤的一个危险因素, 空腹饮酒较伴有进餐的饮酒方式更易造成肝损伤^[12](Ⅲ)。

女性对酒精介导的肝毒性更敏感, 与男性相比, 更小剂量和更短的饮酒期限就可能出现更重的酒精性肝病^[13](Ⅲ)。饮用同等量的酒精饮料, 男女血液中酒精水平有明显差异^[14](Ⅱ-2)。

种族^[15](Ⅱ-2)、遗传^[16-17](Ⅲ)以及个体差异^[8](Ⅲ)也是酒精性肝病的重要危险因素。汉族人群的酒精性肝病易感基因乙醇脱氢酶 (ADH) 2、ADH3 和乙醛脱氢酶 (ALDH) 2 的等位基因频率以及基因型分布不同于西方国家, 可能是中国嗜酒人群和酒精性肝病的发病率低于西方国家的原因之一^[16]。并不是所有的饮酒者都会出现酒精性肝病, 只是发生在一小部分人群中, 表明同一地区群体之间还存在着个体差异^[8]。

酒精性肝病死亡率的上与营养不良的程度相关^[18](Ⅲ)。维生素 A 的缺少或维生素 E 水平的下降, 也可能加重肝损伤^[19](Ⅲ)。富含多不饱和脂肪酸的饮食可促使酒精性肝病的进展, 而饱和脂肪酸对酒精性肝病起到保护作用^[20](Ⅲ)。肥胖或体质量超重可增加酒精性肝病进展的风险^[8](Ⅲ)。

肝炎病毒感染与酒精对肝脏损害起协同作用^[21](Ⅲ), 在肝炎病毒感染基础上饮酒, 或在酒精性肝病基础上并发 HBV 或 HCV 感染, 都可加速肝脏疾病的发生和发展。

三、酒精性肝病临床诊断标准

1. 有长期饮酒史, 一般超过 5 年, 折合乙醇量男性 ≥ 40 g/d, 女性 ≥ 20 g/d, 或 2 周内有大量饮酒史, 折合乙醇

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量 $>80 \text{ g/d}^{[22]}$ 。但应注意性别、遗传易感性等因素的影响。乙醇量 (g) 换算公式 = 饮酒量 (ml) \times 乙醇含量 (%) $\times 0.8$ 。

2. 临床症状为非特异性, 可无症状, 或有右上腹胀痛、食欲不振、乏力、体质量减轻、黄疸等; 随着病情加重, 可有神经精神症状和蜘蛛痣、肝掌等表现^[22](*III*)。

3. 血清天冬氨酸氨基转移酶 (AST) (*II-2*)、丙氨酸氨基转移酶 (ALT) (*III*)、 γ -谷氨酰转肽酶 (GGT) (*II-2*)、总胆红素 (TBil) (*III*)、凝血酶原时间 (PT) (*III*)、平均红细胞容积 (MCV) (*II-2*) 和缺糖转铁蛋白 (CDT) (*II-2*) 等指标升高^[22-25]。其中 AST/ALT >2 、GGT 升高、MCV 升高为酒精性肝病的特点, 而 CDT 测定虽然较特异但临床未常规开展。禁酒后这些指标可明显下降, 通常 4 周内基本恢复正常 (但 GGT 恢复较慢)^[26-27](*II-2*), 有助于诊断。

4. 肝脏 B 超或 CT 检查有典型表现^[28-31](见本《指南》影像学诊断部分)(*II-2*)。

5. 排除嗜肝病毒现症感染以及药物、中毒性肝损伤和自身免疫性肝病等^[22](*III*)。

符合第 1、2、3 项和第 5 项或第 1、2、4 项和第 5 项可诊断酒精性肝病, 仅符合第 1、2 项和第 5 项可疑诊酒精性肝病^[22]。符合第 1 项, 同时有病毒性肝炎现症感染证据者, 可诊断为酒精性肝病伴病毒性肝炎。

符合酒精性肝病临床诊断标准者, 其临床分型诊断如下。

1. 轻度酒精性肝病: 肝脏生物化学指标、影像学和组织病理学检查基本正常或轻微异常。

2. 酒精性脂肪肝: 影像学诊断符合脂肪肝标准, 血清 ALT、AST 或 GGT 可轻微异常。

3. 酒精性肝炎: 是短期内肝细胞大量坏死引起的一组临床病理综合征, 可发生于有或无肝硬化的基础上, 主要表现为血清 ALT、AST 升高和血清 TBil 明显增高, 可伴有发热、外周血中性粒细胞升高。重症酒精性肝炎是指酒精性肝炎患者出现肝功能衰竭的表现, 如凝血机制障碍、黄疸、肝性脑病、急性肾功能衰竭、上消化道出血等, 常伴有内毒素血症。

4. 酒精性肝硬化: 有肝硬化的临床表现和血清生物化学指标的改变。

四、影像学诊断^[28-31]

影像学检查用于反映肝脏脂肪浸润的分布类型, 粗略判断弥漫性脂肪肝的程度, 提示是否存在肝硬化, 但其不能区分单纯性脂肪肝与脂肪性肝炎, 且难以检出 $<33\%$ 的肝细胞脂肪变; 应注意弥漫性肝脏回声增强以及 CT 密度值降低也可见于其他慢性肝病。

(一) 超声显像诊断

具备以下三项腹部超声表现中的两项者为弥漫性脂肪肝: (1) 肝脏近场回声弥漫性增强, 回声强于肾脏; (2) 肝脏远场回声逐渐衰减; (3) 肝内管道结构显示不清。

(二) CT 诊断

弥漫性肝脏密度降低, 肝脏与脾脏的 CT 值之比 ≤ 1 。弥漫性肝脏密度降低, 肝/脾 CT 比值 ≤ 1.0 但大于 0.7 者为轻度; 肝/脾 CT 比值 ≤ 0.7 但 >0.5 者为中度; 肝/脾 CT

比值 ≤ 0.5 者为重度。

五、组织病理学诊断^[22]

酒精性肝病病理学改变主要为大泡性或大泡性为主伴小泡性的混合性肝细胞脂肪变性。依据病变肝组织是否伴有炎症反应和纤维化, 可分为单纯性脂肪肝、酒精性肝炎、肝纤维化和肝硬化。酒精性肝病的病理学诊断报告应包括肝脂肪变程度 (F0~4)、炎症程度 (G0~4)、肝纤维化分级 (S0~4)。

(一) 单纯性脂肪肝

依据脂肪变性肝细胞占肝组织切片的比例, 依据肝细胞脂肪变性占据所获取肝组织标本量的范围, 分为 4 度 (F0~4): F0 $<5\%$ 肝细胞脂肪变; F1 5%~33% 肝细胞脂肪变; F2 33%~66% 肝细胞脂肪变; F3 66%~75% 肝细胞脂肪变; F4 75% 以上肝细胞脂肪变。

(二) 酒精性肝炎和肝纤维化

酒精性肝炎时肝脂肪变程度与单纯性脂肪肝一致, 分为 4 度 (F0~4), 依据炎症程度分为 4 级 (G0~4): G0 无炎症; G1 腺泡 3 带呈现少数气球样肝细胞, 腺泡内散在个别点灶状坏死和中央静脉周围炎; G2 腺泡 3 带明显气球样肝细胞, 腺泡内点灶状坏死增多, 出现 Mallory 小体, 门管区轻至中度炎症; G3 腺泡 3 带广泛的气球样肝细胞, 腺泡内点灶状坏死明显, 出现 Mallory 小体和凋亡小体, 门管区中度炎症伴和(或)门管区周围炎症; G4 融合性坏死和(或)桥接坏死。

依据纤维化的范围和形态, 肝纤维化分为 4 期 (S0~4): S0 无纤维化; S1 腺泡 3 带局灶性或广泛的窦周/细胞周纤维化和中央静脉周围纤维化; S2 纤维化扩展到门管区, 中央静脉周围硬化性玻璃样坏死, 局灶性或广泛的门管区星芒状纤维化; S3 腺泡内广泛纤维化, 局灶性或广泛的桥接纤维化; S4 肝硬化。

酒精性肝病的病理学诊断报告需包括肝脂肪变程度 (F0~4)、炎症程度 (G0~4)、肝纤维化分级 (S0~4)。

3. 肝硬化: 肝小叶结构完全毁损, 代之以假小叶形成和广泛纤维化, 为小结节性肝硬化。根据纤维间隔有否界面性肝炎, 分为活动性和静止性。

六、酒精性肝病的治疗

(一) 评估方法^[32-34](*II-2*)

有多种方法用于评价酒精性肝病的严重程度及近期存活率, 主要包括 Child-Pugh 分级、凝血酶原时间-胆红素判别函数 (Maddrey 判别函数) 以及终末期肝病模型 (MELD) 积分等, 其中 Maddrey 判别函数有较高价值, 其计算公式为: $4.6 \times \text{PT(s)} \text{ 差值} + \text{TBil (mg/dl)}$ 。

(二) 治疗

酒精性肝病的治疗原则是: 戒酒和营养支持, 减轻酒精性肝病的严重程度, 改善已存在的继发性营养不良和对症治疗酒精性肝硬化及其并发症^[35-36]。

1. 戒酒: 戒酒是治疗酒精性肝病最重要的措施^[37](*I*), 戒酒过程中应注意防治戒断综合征。

2. 营养支持: 酒精性肝病者需良好的营养支持, 应在戒酒的基础上提供高蛋白, 低脂饮食, 并注意补充维生素 B、维生素 C、维生素 K 及叶酸^[38](*II-2*)。

3. 药物治疗: (1) 糖皮质激素可改善重症酒精性肝炎

(有脑病者或 Maddrey 指数 > 32) 患者的生存率^[39-40](I)。(2) 美他多辛可加速酒精从血清中清除, 有助于改善酒精中毒症状和行为异常^[41](I)。(3) S-腺苷蛋氨酸治疗可以改善酒精性肝病患者的临床症状和生物化学指标^[42-43](I)。多烯磷脂酰胆碱对酒精性肝病患者有防止组织学恶化的趋势^[44-45](I)。甘草酸制剂、水飞蓟素类、多烯磷脂酰胆碱和还原型谷胱甘肽等药物有不同程度的抗氧化、抗炎、保护肝细胞膜及细胞器等作用, 临床应用可改善肝脏生物化学指标^[44, 46-47](II-2, II-3)。双环醇治疗也可改善酒精性肝损伤^[48](II-2)。但不宜同时应用多种抗炎保肝药物, 以免加重肝脏负担及因药物间相互作用而引起不良反应(III)。(4) 酒精性肝病者肝脏常伴有肝纤维化的病理改变, 故应重视抗肝纤维化治疗(III)。目前有多种抗肝纤维化中成药或方剂, 今后应根据循证医学原理, 按照新药临床研究规范(GCP)进行大样本、随机、双盲临床试验, 并重视肝组织学检查结果, 以客观评估其疗效和安全性。(5) 积极处理酒精性肝硬化的并发症(如门静脉高压、食管胃底静脉曲张、自发性细菌性腹膜炎、肝性脑病和肝细胞肝癌等)^[37](III)。(6) 严重酒精性肝硬化患者可考虑肝移植, 但要求患者肝移植前戒酒 3~6 个月, 并且无其他脏器的严重酒精性损害^[49](II-2)。

主要参与者: 厉有名、范建高、王炳元、陆伦根、施军平、牛俊奇、沈薇、徐有青

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