

· 综述 ·

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无创指标动态变化对代谢相关脂肪性肝病临床结局的评估效能

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摘要: 病理组织学检查是目前诊断代谢相关脂肪性肝病(MAFLD)的金标准,然而其为有创操作,存在风险且可行性低,因此利用无创指标评估MAFLD分期和分型成为当前研究热点。本文系统综述了不同无创指标的动态变化在反映MAFLD患者肝组织学改变及临床终点事件方面的效能。

关键词: 非酒精性脂肪性肝病; 生物标记; 诊断显像; 模型, 统计学

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Efficacy of the dynamic changes of noninvasive indicators in evaluating clinical outcomes of metabolic associated fatty liver disease

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Abstract: Histopathological examination is currently the gold standard for the diagnosis of metabolic associated fatty liver disease (MAFLD); however, due to its invasiveness, high risks, and low feasibility, application of noninvasive indicators in the staging and classification of MAFLD has become a research hotspot. This article systematically reviews the efficacy of dynamic changes in various noninvasive markers in reflecting histological changes and clinical outcome events in MAFLD patients, in order to provide theoretical support for dynamic monitoring and individualized management of the disease.

Key words: Non-alcoholic Fatty Liver Disease; Biomarkers; Diagnostic Imaging; Models, Statistical

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代谢相关脂肪性肝病(metabolic associated fatty liver disease, MAFLD),既往称为非酒精性脂肪性肝病(non-alcoholic fatty liver disease, NAFLD),是一类与遗传易感性、营养过剩和代谢紊乱等因素相关的慢性进展性肝病^[1]。近年来,全球MAFLD发病率和患病率逐年升高,我国总患病率亦增长至近30%^[2]。MAFLD可进展为代谢相关脂肪性肝炎(metabolic associated steatohepatitis, MASH)、代谢相关脂肪性肝纤维化和肝硬化,甚至发生肝脏相关终点事件。目前,肝穿刺活组织检查仍是MAFLD分期及分型的金标准^[1],但在临床实践中,肝穿刺为有创操作,不易普及且有一定风险。

因此,探索可用于替代肝穿刺诊断及评估疾病进展的无创指标成为当前的研究热点。尽管已有部分无创指标可用于评估MAFLD患者脂肪变性、炎症和纤维化程度,但是其动态变化是否能反映组织学改变及肝脏相关终点事件的发生,仍有待进一步验证。

1 无创指标动态变化可评估组织学改变

在MAFLD的组织学评估中,相关评价指标主要有NAFLD活动度评分(NAFLD activity score, NAS)及纤维化分期。目前MASH相关的新药研究多以组织学改善即MASH缓解和纤维化逆转为主要疗效评价指标^[3],部

分新药研究中,将一些无创指标变化纳入探索性终点,但这些无创指标能否作为替代终点仍需进一步研究。

1.1 部分血清学无创指标变化可一定程度反映组织学改变

1.1.1 天冬氨酸氨基转移酶和血小板比率指数(AST to platelet ratio index, APRI)、肝纤维化4项(fibrosis-4, FIB-4)和NAFLD纤维化评分(NAFLD fibrosis score, NFS)动态变化与纤维化改变的关系有待验证 APRI、FIB-4和NFS是基于临床常规检验指标构建的无创模型,常用于MAFLD相关纤维化的诊断。近年研究表明,APRI、FIB-4、NFS对患者进展为晚期纤维化(\geq F3)具有较好的预测能力,其中APRI动态变化在评估纤维化改变中的效能优于FIB-4、NFS,但这3项指标的动态变化与纤维化改变相关性均较弱(回归系数分别为0.33、0.26和0.19)^[4]。

1.1.2 增强型肝纤维化(enhanced liver fibrosis, ELF)评分及其相关标志物水平变化与组织学改变有相关性 ELF评分由透明质酸(hyaluronic acid, HA)、金属蛋白酶组织抑制因子I(tissue inhibitor of matrix metalloproteinase 1, TIMP-1)和Ⅲ型胶原蛋白前肽(procollagen Ⅲ amino-terminal peptide, PⅢNP)定量水平计算所得,对显著纤维化和肝硬化具有良好的诊断价值^[5]。

ELF评分及其相关指标的下降可在一定程度上反映MASH组织学炎症、脂变及纤维化的改善。PIVENS试验结果表明,非肝硬化MASH患者接受维生素E或吡咯酮治疗96周时,ELF评分下降与NAS评分改善显著相关[比值比(odds ratio, OR)=1.59, 95%置信区间(confidence interval, CI):1.03~2.45, $P=0.04$]^[6]。其中, PⅢNP的动态变化可较好反映纤维化分期改变,在治疗96周时PⅢNP水平下降与纤维化逆转相关性较高($OR=1.23$, $95\%CI: 1.07 \sim 1.42$, $P=0.004$)^[6]。此外,作为Ⅲ型胶原的另一个标志物, N-末端Ⅲ型胶原蛋白前肽(N-terminal type 3 collagen propeptide, Pro-C3)^[7]的减少可提示组织学改善。非肝硬化MASH患者经NGM282治疗12周后,出现组织学改善患者的Pro-C3水平较基线下降率为56%,是未改善患者的6.2倍^[8]。

1.1.3 细胞角蛋白18片段(cytokeratin-18, CK18)及相关无创模型可反映组织学改善 CK18是反映MAFLD相关肝细胞死亡的标志物。研究显示,显著纤维化(F2/F3)的MASH患者接受Selonsertib治疗24周后,CK18 M30和CK18 M65的下降与组织学纤维化逆转有相关性(CK18 M30: $OR=1.25$, $95\%CI: 1.04 \sim 1.52$, $P=0.02$; CK18

M65: $OR=1.12$, $95\%CI: 1.00 \sim 1.25$, $P=0.04$)^[9]。此外,由CK18 M30、趋化因子配体10(CXCL10)和体重指数组成的N3-MASH模型,也被证实可用于评估MASH的改善情况。研究发现, N3-MASH评分在发生MASH组织学改善的患者中显著降低^[10]。

1.2 影像学无创指标变化可评估组织学改变

1.2.1 磁共振质子密度脂肪分数(magnetic resonance imaging-derived proton density fat fraction, MRI-PDFF)下降最适监测MASH缓解,与纤维化逆转相关性有待验证 在脂肪变性与炎症反应方面,基于FLINT试验的一项研究发现, MRI-PDFF相对下降30%是反映NAS评分改善的最佳阈值^[11]。一项Meta分析进一步证实, MRI-PDFF相对下降30%的患者更易发生MASH缓解($OR=5.45$, $95\%CI: 1.53 \sim 19.46$, $P=0.009$)^[12]。目前,治疗后MRI-PDFF较基线的变化,已成为部分新药Ⅱ期临床试验的主要研究终点或次要研究终点^[13-14],提示MRI-PDFF在监测肝脂肪变性与炎症动态变化方面具有显著优势。

在肝纤维化改善方面,一项纳入100例配对肝穿刺活组织检查和MRI-PDFF检查的研究表明, MRI-PDFF相对下降 $\geq 30\%$ 是肝纤维化逆转的独立预测因素($OR=6.46$, $95\%CI: 1.1 \sim 37.0$, $P=0.04$)^[15]。但该研究中, MRI-PDFF下降的患者中有近37.6%的患者肝组织学纤维化分期未改善甚至进展,这可能与随访时间短(仅1.5年)、脂肪变性减少与纤维化逆转的发生不同步,以及MRI-PDFF测量脂肪含量不能直接反映纤维化变化等因素有关。未来仍需在干预措施下进行长期随访研究,验证MRI-PDFF变化与肝纤维化改变的关系。

尽管MRI-PDFF在评估肝脂肪变性与炎症反应方面已得到普遍认可,但目前尚无指南推荐用血清学或影像学指标动态监测MAFLD患者组织学变化,肝穿刺活组织检查仍是评价肝脂肪变性、纤维化动态变化的金标准。

1.2.2 瞬时弹性成像(transient elastography, TE)及磁共振弹性成像(magnetic resonance elastography, MRE)可较准确评估肝纤维化变化 肝脏TE是一项评估肝硬度值(liver stiffness measurement, LSM)的无创技术,在临床中广泛应用。一项随访10年的研究发现, TE-LSM的改变与MAFLD患者纤维化分期改变相关($r=0.56$, $P=0.036$)^[16]。另一项研究也证实,奥贝胆酸治疗18个月后,纤维化逆转的患者TE-LSM下降率为10.9%~19.8%,但在肝纤维化分期不变的患者中, TE-LSM也下

降了7.6%^[17],提示存在假阳性结果。这种假阳性结果可能与血清丙氨酸氨基转移酶(alanine aminotransferase, ALT)水平下降、肝脂肪变性或炎症缓解、患者体重明显下降等因素相关^[18]。由于TE-LSM易受多种非肝纤维化因素干扰,其在判断肝纤维化逆转方面的特异性有限,因此需结合组织学或其他生物标志物综合评估。

MRE在诊断MAFLD患者肝纤维化分期中的准确性较高^[19]。MRE的动态变化也与肝纤维化分期改变有关。一项纳入102例配对肝穿刺活组织检查和MRE检查的研究表明,MRE增加15%是发生晚期肝纤维化(\geq F3)较好的预测指标($OR=4.90, 95\%CI: 1.35 \sim 17.84, P=0.016$)^[20]。然而,MRE的预测效能较低,MASH患者Selonsertib治疗24周后MRE增加 $\geq 15\%$ 预测肝纤维化进展的敏感度仅为13%,特异度为72%^[21]。因此,MRE能否较好且稳定地评估MASH纤维化动态改变仍需长时间、多样本的临床研究验证,并探索MRE与其他无创指标联合以期提高预测效能。

1.3 血清学联合影像学模型变化能较好监测MASH缓解 FAST评分是由FibroScan测得的TE-LSM、受控衰减参数(controlled attenuation parameter, CAP)和血清天冬氨酸氨基转移酶(aspartate aminotransferase, AST)3种指标构成,可识别高风险MASH患者。一项全球多中心司美格鲁肽IIb期临床研究结果表明,治疗72周后,FAST评分下降可预测MASH缓解,受试者操作特征曲线下面积(area under curve, AUC)为0.69(95%CI: 0.58~0.81)^[22]。然而,在肝纤维化逆转的患者中,FAST评分的改变没有统计学意义。因此,FAST评分是否能作为潜在的组织学替代终点,仍需更多长期随访研究验证。

为进一步精准评估MASH疾病进展情况,Loomba等^[23]利用ALT变化值、MRI-PDFF变化值及基线时AST水平,构建了MASH缓解指数(MASH resolution index, MASHResInd)模型,该模型在预测MASH缓解方面具有良好的预测效能,建模组的AUC为0.81(95%CI: 0.69~0.93),验证组的AUC为0.83(95%CI: 0.76~0.91)。

一项针对160例F2~F3期MAFLD患者行头对头的比较研究显示,MASHResInd对应的AUC显著高于FAST评分改变[AUC: 0.83(0.75~0.91) vs 0.65(0.55~0.75), $P=0.001$],且MASHResInd阳性预测值、阴性预测值均高于FAST评分,提示MASHResInd在预测MASH缓解方面具有更优的性能^[24]。

2 无创指标动态变化评估远期终点事件仍需更多证据

MAFLD患者发生肝硬化失代偿和肝细胞癌的风险较高,且随纤维化进展进一步增加^[25]。无创指标可预测MASH患者发生肝脏相关事件(liver-related event, LRE)的风险,其动态变化也与MASH远期临床结局发生有一定关联。

在血清学方面,有研究表明FIB-4、NFS的变化与LRE发生有关。一项对代偿期肝硬化(F4)MASH患者随访2年的研究表明,FIB-4评分升高是发生LRE的危险因素[风险比(hazard ratio, HR)=1.10, 95%CI: 1.01~1.21, $P=0.040$]^[26]。另有研究指出,末次FIB-4水平较其变化值与发生LRE的相关性更强^[27],提示动态监测FIB-4对临床预后评估具有重要意义。此外,NFS的升高与第1次发生LRE相关($HR=1.66, 95\%CI: 1.23 \sim 2.25, P=0.001$)^[28]。在预测效能方面,一项针对135例MASH患者平均随访12.6年的研究显示,APRI、FIB-4的变化对预测终末期肝病的效能较好,NFS则欠佳(AUC: 0.73 vs 0.80 vs 0.58)^[29]。

TE-LSM对预测MAFLD相关终点事件发生的影像学价值备受关注。研究表明,TE-LSM的动态变化是预测失代偿期肝硬化、肝细胞癌及肝脏相关死亡事件发生的独立危险因素^[30]。一项针对2508例全病因慢性肝病患者(MAFLD占15%)的研究表明,LSM增加20%与失代偿风险增加约50%相关($HR=1.45, 95\%CI: 1.41 \sim 1.79, P<0.001$),且LSM的动态变化对随后1年失代偿的发生预测效能较高,AUC为0.933,优于FIB-4动态变化、MELD评分及单独两次测量的LSM^[31]。Liu等^[32]对中位随访55个月的MAFLD患者多次测量CAP及LSM,并对二者进行轨迹分析,发现部分晚期肝纤维化患者在随访过程中出现CAP水平迅速下降、LSM持续升高的现象,并将这部分患者定义为“burning-out”MAFLD。这群患者全因死亡、LRE和肝硬化失代偿10年累积发生率均较高,提示临床医生在评估晚期MAFLD进展时需同时动态监测肝脂肪变性及纤维化程度。此外,MRE的动态变化也与临床结局的发生有关。一项研究表明,发生LRE的MAFLD患者,MRE平均每年增加0.42 kPa,且MRE进展的患者(定义为中位随访时间7.7年,MRE年增幅为0.61 kPa)发生失代偿或死亡的风险是非进展患者的19倍^[33]。目前,仅针对MAFLD患者设计的、与影像学相关且观察临床结局变化的研究较少,未来仍需较长时间、更多样本的临床研究进一步验证。

血清联合影像的无创模型 Agile 3+、Agile 4 包含 AST/ALT 比值、血小板计数及 TE-LSM 等参数,可分别诊断 MAFLD 患者进展期肝纤维化和肝硬化。一项全球多中心研究结果显示,基线时高 Agile 3+ 评分的患者, Agile 3+ 下降 $\geq 20\%$ 可显著降低 LRE 发生风险^[34],但在基线低或中间水平 Agile 3+ 评分的患者中未观察到与临床结局的相关性,未来仍需更多临床研究进一步探索。

3 小结

当前,越来越多的研究表明,无创指标变化与 MAFLD 组织学改变及终点事件结局的发生有关,但其在预测临床结局方面的准确性仍有待提高。目前尚无临床指南推荐无创指标动态变化可替代肝穿刺活组织检查评估肝脂肪变性、纤维化变化及临床终点结局^[18,35-36]。因此,未来研究需建立更有效的无创指标模型或优化现有无创指标组合,以提升其在疾病动态监测与结局预测中的价值,为替代肝穿刺活组织检查实现精准无创的疾病评估提供依据。

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