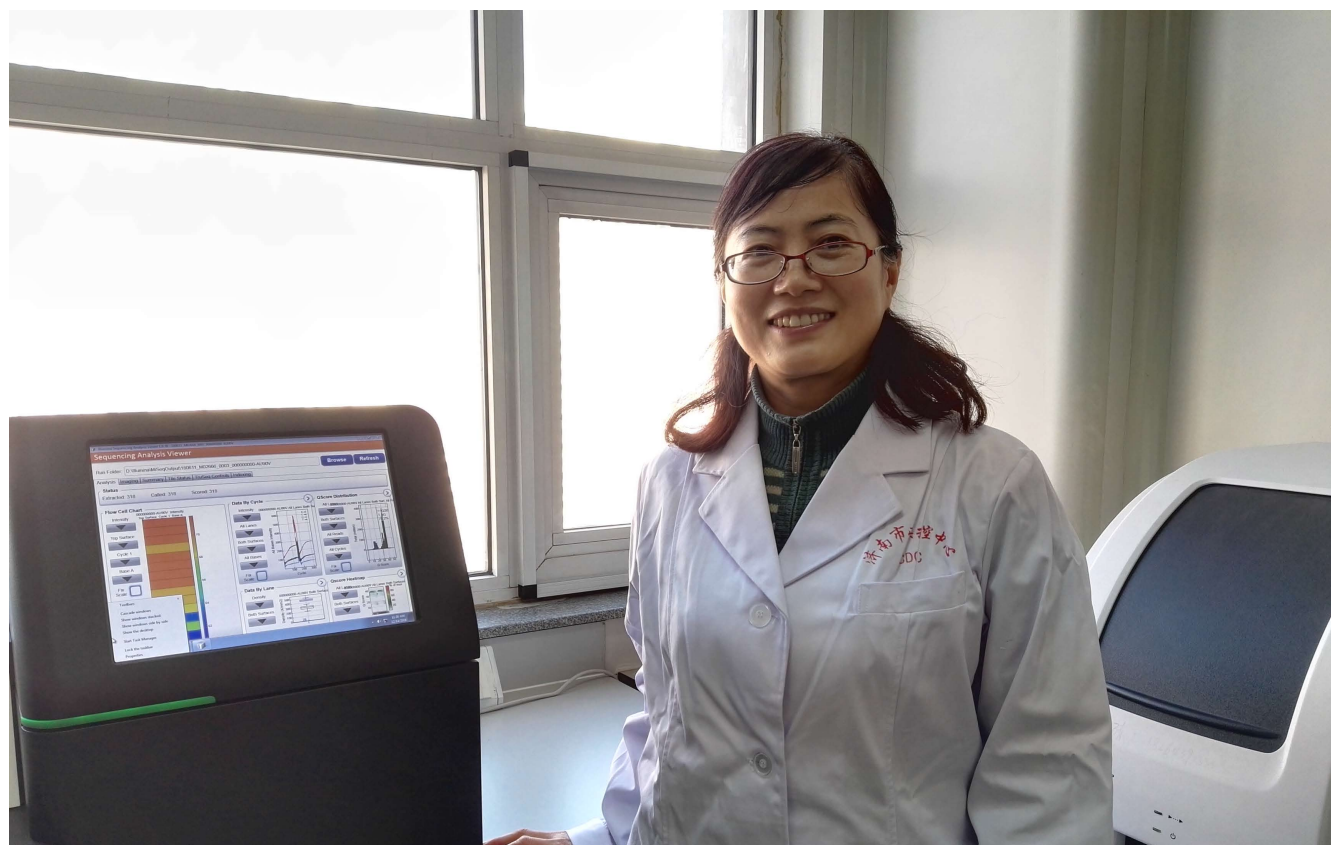


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肝窦阻塞综合征的影像学表现

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Imaging manifestations of hepatic sinusoidal obstruction syndrome

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Abstract

Hepatic sinusoidal obstruction syndrome (HSOS), previously known as hepatic veno-occlusive disease, is a kind of vascular disorder of the liver. HSOS is caused

by toxic destruction of hepatic sinusoidal endothelial cells. Major etiologies of HSOS include hematopoietic stem cell transplantation, oxaliplatin-containing chemotherapy, intake of pyrrolizidine alkaloid-containing herbal remedies, etc. There have been many studies on the imaging manifestations of HSOS in recent years. This paper summarizes the imaging manifestations of HSOS by reviewing the latest literature and our work.

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Key Words: Hepatic sinusoidal obstruction syndrome; Pyrrolizidine alkaloid; Hematopoietic stem cell transplantation; Imaging

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摘要

肝窦阻塞综合征(hepatic sinusoidal obstruction syndrome, HSOS), 又称肝小静脉闭塞病, 是由各种原因导致肝窦内皮细胞损害致肝窦流出道阻塞所引起的肝脏血管性疾病, 其主要的病因包括: 造血干细胞移植、使用奥沙利铂等化疗药物、服用含吡咯烷生物碱的中草药等. 近几年关于HSOS影像学特征的研究不断深入, 本文结合最新国内外文献和本研究组的工作, 将HSOS影像学特征做一总结.

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关键词: 肝窦阻塞综合征; 吡咯烷生物碱; 造血干细胞移植; 影像学

核心提要: 肝窦阻塞综合征(hepatic sinusoidal obstruction syndrome, HSOS)是由各种原因导致肝窦内皮细胞损害致肝窦流出道阻塞所引起的肝脏血管性疾病, 本文结合最新国内外文献和本研究组的工作, 阐述HSOS影像学特征。

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0 引言

肝窦阻塞综合征(hepatic sinusoidal obstruction syndrome, HSOS)的发病机制为各种原因造成肝窦内皮细胞/中央静脉内皮细胞损伤、脱落, 肝窦扩张充血; 肝细胞肿胀、坏死, 红细胞渗入Disse间隙, 进而肝内小静脉管壁增厚, 管腔狭窄、闭塞^[1-3]。目前, HSOS常见的致病原因有三种: (1)造血干细胞移植(hematopoietic stem cell transplantation, HSCT); (2)应用抗肿瘤化疗药如奥沙利铂等; (3)服用含吡咯烷生物碱的中草药如土三七等^[2-4]。欧美报道的病例多为HSCT后。在我国, 服用含吡咯烷生物碱的中草药(主要为土三七)而起病者占多数^[1,2,4]。

肝窦阻塞综合征病情较重, 目前缺乏有效的治疗手段, 病死率较高。肝窦阻塞综合征在临床上较为少见, 目前临床上常用的诊断标准为西雅图和巴尔的摩诊断标准, 其主要包含服药或HSCT的病史、黄疸、肝肿大或右上腹痛、腹水、体重增加, 但是以上诊断标准以临床表现为依据, 缺乏特异性^[4-6]。肝穿刺活检是本病诊断的金标准, 但此类患者常常因血小板减少、凝血功能障碍、大量腹腔积液等, 而无法进行经皮肝穿刺活检; 经静脉肝穿刺活检在很多地方因设备和费用问题而运用受限。此外, HSOS患者的肝脏病变呈斑片状分布, 这一特点可能会导致肝穿刺病理出现假阴性^[4,7]。在肝脏疾病诊断中, 影像学检查, 尤其是超声、CT和MRI能提供有效的诊断价值。本文结合最新国内外文献和本研究组的工作, 对于不同病因肝窦阻塞综合征的影像学表现作如下综述, 以提高临床医生对肝窦阻塞综合征影像学特征的认识, 从而为其诊断和治疗提供帮助。

1 吡咯烷生物碱诱导的肝窦阻塞综合征的影像学特征

服用含吡咯烷生物碱的中草药是我国HSOS的最主要的病因, 其中最常用的中药为土三七^[8-10]。该病临床症状缺乏特异性, 诊断需要吡咯烷生物碱服用史^[11]。全世界大约有3%的开花植物含吡咯烷生物碱, 且传统中药处方由多种药物构成^[12], 故难以确定HSOS患者吡

咯烷生物碱的服用情况。近年来, 复旦大学王吉耀等利用超高效液相色谱-质谱法(Ultra performance liquid chromatography-mass spectrometry, UPLC-MS)可以检测吡咯烷生物碱导致的HSOS患者血液中的吡咯-蛋白加合物, 其可作为吡咯烷生物碱导致HSOS的生物标记物^[9]。但UPLC-MS在国内大多数医院未广泛开展, 且患者血液中吡咯-蛋白加合物浓度随时间而逐渐下降, 检测结果受检测时机影响较大。因此, 需要一种无创的方法来辅助诊断吡咯烷生物碱导致的HSOS, 影像学是常见的无创检查, 对疾病的诊断意义较高。

1.1 超声特征 南京鼓楼医院的诸葛宇征等发现肝肿大, 门静脉血流速度降低为吡咯烷生物碱导致HSOS的二维超声的重要特征^[13]。2017年中国专家共识意见提示: 二维超声的典型表现包括: 肝脏弥漫性肿大; 肝实质回声增粗增密, 分布不均匀, 可见沿肝静脉走行的“斑片状”回声减低区; 腹腔积液。彩色多普勒超声的表现是门静脉、脾静脉内径正常, 血流速度减慢(<25 cm/s)。超声造影的表现是动脉期呈“花斑样”不均匀增强; 门静脉充盈缓慢; 肝动脉-肝静脉渡越时间延长^[1]。目前, 关于超声的研究较少, 影像特征缺乏特异性, 因此, 超声的诊断价值有限。

1.2 CT特征 本研究小组在2016年的研究发现: 病人CT多表现为腹水, 肝肿大, 胆囊壁增厚, 胸腔积液, 肝静脉狭窄, 肝脏斑片状强化, 以及不均匀低密度病灶, 其中以肝脏斑片状强化, 不均匀低密度病灶为其特征性CT表现^[14]。CT的诊断价值明显优于西雅图诊断标准中各个参数^[14]。南京鼓楼医院的诸葛宇征的研究表明: CT表现包括肝肿大, 腹水, 密度不均匀或“地图状”增强(平衡阶段的不均匀增强), 肝静脉模糊不清; 脾肿大、食管和胃底静脉曲张发生率低^[13]。温州医学院的Shao等^[15]的研究发现腹水、肝实质密度不均匀, 门静脉期低密度区为共同表现。2017年中国专家共识意见提示: (1)肝脏弥漫性肿大, 平扫显示肝实质密度不均匀减低; (2)静脉期和平衡期肝实质呈特征性“地图状”、“花斑样”不均匀强化, 门静脉周围出现的低密度水肿带称为“晕征”; (3)尾状叶、肝左外叶受累稍轻, 肝静脉周围肝实质强化程度较高, 呈现特征性“三叶草征”, 肝静脉管腔狭窄或显示不清, 下腔静脉肝段受压变细; (4)通常合并腹水、胸水、胆囊壁水肿和胃肠壁水肿等肝外征象^[1]。此外, Wu等^[16]和Zhou等^[17]也证实类似CT的特征。综上所述, 不均匀低密度灶、地图样或斑片样强化为吡咯烷生物碱造成HSOS相对特异的CT特征。

1.3 MRI特征 本研究小组2017年研究结果显示: 病人多表现为腹水, 肝肿大, 胆囊壁水肿, 门脉区水肿, 胸腔积液, 肝脏不均一低信号病变, 肝右静脉狭窄, 肝右静脉显

影不清, 下腔静脉狭窄等. 在上述表现中, 门脉期的肝脏不均一低信号病变是特征性表现, 大多数表现为沿肝静脉分布的“爪形”不均一强化, 病变区域在SWI和T2*序列上信号显著减低^[18]. 浙江大学第一附属医院的Zhou等^[17]的研究显示: MRI均表现为腹水, 斑片状肝脏增强, 右主肝静脉狭窄或闭塞, 肝肿大和胆囊壁增厚. 2017年中国专家共识意见提示: 平扫表现为肝脏体积增大和大量腹水, 肝脏信号不均, 3支肝静脉纤细或显示不清; T2加权成像(T2WI)表现为片状高信号, 呈“云絮”状. MRI动态增强扫描表现为动静脉期不均匀强化, 呈“花斑”状, 延迟期强化更明显^[1]. 这些均表明: 吡咯烷生物碱造成HSOS的MRI与CT特征具有高度的一致性: 不均一低信号/低密度MRI: 低信号CT: 低密度及不规则强化为相对特异性表现. 因此, MRI及CT对于吡咯烷生物碱造成的HSOS的诊断价值较高.

2 HSCT后的肝窦阻塞综合征的影像学特征

HSCT近几十年来常用于血液疾病与肿瘤疾病的治疗. HSCT术后常会出现肝窦阻塞综合征和移植物抗宿主病等并发症, 而肝窦阻塞综合征及肝脏移植物抗宿主病的临床表现缺乏特异性, 诊断较为困难^[5,19]. 肝脏穿刺活检不宜进行, 此时, 无创的影像学检查对疾病有较好的诊断意义.

2.1 超声特征 McCarville等的研究表明, 门静脉血流速度的降低是HSCT相关HSOS的特征, 但是诊断价值不及西雅图标准^[20]. 此外, Lassau等^[21]与Zieger等^[22]的研究也表明, 超声的诊断价值灵敏性及特异性不高. Yoshimoto等^[23]的研究显示, 灰阶超声表现包括腹水、肝肿大、胆囊壁增厚、肝静脉狭窄等, 这些表现特异性不高; 彩色多普勒超声对门静脉分支的逆行血流的检测对肝窦阻塞综合征有早期诊断价值. 以上这些表明: 超声影像对于HSCT后的肝窦阻塞的诊断价值存在争议, 诊断价值有待进一步研究证实.

2.2 CT特征 Erturk等^[24]比较HSCT后的肝窦阻塞综合征与移植物抗宿主病的CT表现发现: 肝窦阻塞综合征的CT表现主要为腹水、门静脉水肿, 右肝静脉直径小; 而移植物抗宿主病较少出现腹水、门静脉水肿, 小肠壁水肿为特征性表现, 右肝静脉直径较大(平均值为0.87 cm), 这为两者的鉴别诊断提供帮助.

2.3 MRI特征 Bosch等^[25]的病例报告显示: HSCT造成的肝窦阻塞综合征MRI表现为肝肿大, 肝静脉狭窄, 门静脉周围狭窄, 胆囊壁增厚, T2加权胆囊壁明显高信号, 腹水. 其中胆囊壁增厚, 信号均匀, 高信号, T2加权胆囊壁明显高信号为其特征性表现. 目前, 关于HSCT后的HSOS的MRI表现的研究较少, 诊断价值有限.

3 化疗药物(如奥沙利铂)造成肝窦阻塞综合征的影像学特征

奥沙利铂为第三代铂类化合物, 常用于结直肠癌肝转移的辅助化疗. 肝窦阻塞综合征是结直肠癌全身化疗的不良反应之一, 其主要是由奥沙利铂造成肝窦内皮细胞损伤进而导致HSOS. 奥沙利铂造成的肝窦阻塞综合征可能无临床症状, 但是, 它会增加肝切除术后肝功能衰竭的发生率^[26-28]. 因此, 疾病的早期诊断尤为重要. 目前, 关于超声影像的研究甚微.

3.1 CT特征 根据Han等^[29]的报道, 奥沙利铂造成HSOS的CT特征为肝实质密度不均一减低, 可呈外部分布、多灶分布、弥漫分布. 本课题组发现全球第一例接受奥沙利铂化疗的胃癌患者发生肝窦阻塞综合征, 其增强CT表现为肝实质密度不均一, 病变表现为不均一的低密度^[30]. 由此可见, 三种常见病因的HSOS的CT表现虽不尽相同, 但均表现为不均一低密度.

3.2 MRI特征 增强MRI肝脏表现为信号强度的不均一性, 增强期的图像显示在肝窦扩张的区域表现为信号强度减弱, 肝胆期网状低信号是结肠癌肝转移使用奥沙利铂造成肝窦阻塞综合征的特征; 诊断具有高度特异性. 但在病变轻微的HSOS患者可能会漏诊^[31-33]. 超顺磁性氧化铁(superparamagnetic iron oxide, SPIO)是一种特殊的MRI造影剂, 其能够选择性被Kupffer枯否细胞选择性的摄取. 在超顺磁性氧化铁增强T2加权梯度回波MRI图像中, 其表现为不同程度的网格状高信号, 在中重度SPIO-增强MRI, MRI诊断敏感性87%, 特异性89%, 阳性预测值83%, 阴性预测值92%^[7]. 但是奥沙利铂在其他肿瘤能否造成HSOS, 国内外尚未见类似报道. 本课题组发现, 在胃癌患者使用奥沙利铂造成的肝窦阻塞综合征中, T2信号不均匀增高, 增强动脉期及延迟期强化欠均匀肝实质密度, 信号不均一减低是其重要的MRI特征^[30].

4 其他原因的肝窦阻塞综合征

在其他病因HSOS中, 使用一种肛交中消遣性药物(popper)可以造成肝窦阻塞综合征, 在MRI平扫期表现为肝脏信号强度不均一, 主要表现为低信号. 下腔静脉通畅. 门脉期: 肝实质不均一强化, 下腔静脉狭窄. 延迟期肝右和肝中静脉狭窄但是通畅^[34]. Shen等^[35]的研究中, 27岁男性患者肝移植术后使用他克莫司后, 出现HSOS, 其CT的特征为: 肝脏增大、斑片样强化、肝静脉显示不清, 大量腹水; 经过120 d治疗后CT显示肝脏基本恢复正常.

5 结论

肝窦阻塞综合征可由多种原因造成, 本文系统收集了国

内外关于不同病因的肝窦阻塞综合征的CT、MRI、超声表现的报道,发现影像学对肝窦阻塞综合征的诊断有较好的辅助价值。本文旨在为临床医师提供参考,从而为肝窦阻塞综合征的诊断和治疗提供新的机遇。

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