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文献综述 REVIEW

原发性小肠淋巴瘤临床病理特点及诊治进展

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Clinicopathological features and treatment of primary small intestinal lymphoma: recent advances

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Abstract
Primary small intestinal lymphoma (PSIL) is an extra-nodal lymphoma whose clinical and histological presentations are usually heterogeneous depending on the site of the lesion. Proper staging criteria are important for clinicopathological diagnosis. Although there is no consensus regarding the role of surgery and chemotherapy in the treatment of PSIL, surgery followed by chemotherapy and radiotherapy is still the main treatment. This review summarizes the clinicopathological features, diagnosis, therapy and prognosis of PSIL.

Key Words: Small intestinal tumors; Lymphoma; Diagnosis; Therapy

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摘要
原发性小肠淋巴瘤(primary small intestinal lymphoma, PSIL)是一种结外淋巴瘤,其临床表现缺乏特异性, 其组织分型、病理形态与结内淋巴瘤有所不同, 临床易误诊漏诊, 病理诊断较困难, 明确其分类、分期标准有助于提高临床及病理诊断水平. 其治疗与结内淋巴瘤及其他胃肠道肿瘤不同, 目前采取以手术为主以放化疗为辅的综合治疗. 本文对该病的临床病理特点、分型、诊治及预后进行综述.

关键词: 小肠肿瘤; 淋巴瘤; 诊断; 治疗

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■背景资料
小肠肿瘤较其他胃肠道肿瘤发病率低, 原发性小肠淋巴瘤是最常见的小肠肿瘤, 在原发性胃肠道淋巴瘤中仅次于胃淋巴瘤, 位居第二. 随着肿瘤发病率的上升, 以及诊疗技术的提高, 小肠淋巴瘤的发病率及检出率都大大提高.

0 引言
原发性小肠淋巴瘤即指发生于淋巴结外的肠道原发性恶性淋巴瘤, 来源于肠壁黏膜下淋巴组织. 近年来由于结外淋巴瘤的发生率呈上升趋势, 胃肠道淋巴瘤发病率也相应增加, 相关因素有病毒感染[1], 如乙型肝炎病毒(hepatitis B virus, HBV)、EB病毒、幽门螺杆菌(*Helicobacter pylori*, *H.pylori*)[2,3]、人类免疫缺陷病毒(human immunodeficiency virus, HIV)[4,5]; 免疫性疾病如Hasmoto's甲状腺炎[6]; 免疫功能缺陷或异常, 如获得性免疫缺陷综合征(acquired immune deficiency syndrome, AIDS), 免疫抑制剂大量使用、放化疗后等[7]; 其他, 如炎症性肠病、腹泻病[8]对其发病率均有影响. 目前随着影像技术和分子生物学检查技术的发展与提高, 小肠淋巴瘤的诊断和治疗受到人们广泛关注. 本文就小肠淋巴瘤的临床病理特点及分期、诊治及预后进行综述.

1 临床表现与分期
PSIL占原发性胃肠道淋巴瘤的20-30%, 可发生于任何年龄, 以成年人多见, 男性多于女性, 好发于回肠[9](60%-65%), 其次是空肠(20%-25%), 十二指肠(6%-8%), 其他(8%-9%)[10,11]. 其临床表现缺乏特异性, 常以腹痛为主要表现, 可伴有腹部不适、腹胀、腹部包块、出血、肠穿孔[12,13]、恶

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■研发前沿
原发性小肠淋巴瘤的诊断、治疗方法尚无统一标准. 找到小肠淋巴瘤独有的特点从而指导制定合理的分期分类, 从分子生物、细胞免疫等方面寻求针对此病的特异性检查方法及治疗手段是当前研究的热点及方向.

表 1 PGIL巴黎分期 (Paris staging system)分期 (2003年)

分期	结果	分期	结果
Tx	淋巴瘤范围不能确定	Nx	淋巴结侵犯不能确定
T0	无淋巴瘤证据	N0	无淋巴结侵犯证据
T1	淋巴瘤局限在黏膜/黏膜下层	N1	侵犯局部淋巴结
T1m	淋巴瘤局限在黏膜	N2	侵犯腹腔内淋巴结, 远处与局部淋巴结
T1sm	淋巴瘤局限在黏膜下层	N3	播散到腹腔外淋巴结
T2	淋巴瘤侵犯肌层或浆膜下层		
T3	淋巴瘤穿透浆膜, 但没有侵及邻近脏器		
T4	淋巴瘤侵及邻近脏器		
Mx	远处播散淋巴瘤不能确定	x	侵犯骨髓不能确定
M0	无结外播散证据	0	无骨髓侵犯证据
M1	不连续的胃肠道不同部位的病变	1	病变侵及骨髓
M2	不连续的其他组织和脏器的病变		
TNMB	临床分期: 肿瘤状态, 淋巴结, 转移, 骨髓		
pTNMB	组织病理学分期: 肿瘤状态, 淋巴结, 转移, 骨髓		
pN	组织学检查通常包括6个或更多淋巴结		

心、呕吐、腹泻、黑便等其他表现, 也可伴有发热、消瘦、食欲下降等全身症状, 同结内淋巴瘤[14]. 有时因淋巴瘤的肿块引起肠套叠诱发急性腹痛[15-17], 若发生在回盲部极易与阑尾炎混淆, 常需急诊手术后明确诊断, 故临床上遇到无明显原因的腹痛、消化道出血、腹部包块和肠梗阻时, 应警惕小肠淋巴瘤的可能, 避免误诊或延迟诊断[18].

临床分期对PGIL的治疗方式选择和预后评估有重要意义. 胃肠道淋巴瘤常需考虑肿瘤浸润深度、淋巴结转移、周围脏器受累等因素, 故被普遍应用的结内淋巴瘤. Ann Arbor分期标准不太适合PSIL. 然而目前国际上对胃肠道淋巴瘤的临床分期也不一致. 2003年提出巴黎分期(paris staging system)[19](表1), 他使用了肿瘤分期通用的TNM方法, 其容易理解且更适用于临床, 有利于对该病的治疗及预后分析.

2 诊断

2.1 临床病理诊断 PSIL临床表现无特异性, 临床诊断较困难, 必须通过各种实验室检查及影像学检查确定病变累及的部位及范围, 即所累及的淋巴结及结外器官, 以协助诊断及临床分期.诊断满足淋巴瘤Dawson标准[20]: (1)无病理性浅表淋巴结肿大; (2)胸片无纵隔淋巴结肿大; (3)末梢血中无幼稚细胞或异常细胞; (4)肿瘤位于小肠或经淋巴管侵犯附近的淋巴结; (5)肝、脾未受侵犯.

病理诊断手段包括显微镜下观察经传统HE染色后的细胞形态特征, 免疫组织化学标记CD3e、CD79a等指标进行免疫分型, 通过聚合酶链反应(PCR)测试B/T细胞的克隆形成能力[21]. PSIL区别于其他胃肠道疾病, 尤其是炎症性肠病的组织学特点是淋巴细胞浸润, 上皮细胞趋性(癌巢或斑块), 细胞核大小不均等[22]. PSIL组织学类型较其他胃肠道淋巴瘤多, 90% PSIL是B细胞来源, 最常见的是弥漫大B细胞淋巴瘤[23], 极少部分是T细胞淋巴瘤或霍奇金淋巴瘤. PSIL的病理诊断即确定是否是淋巴瘤以及其组织学类型[24]. 不同组织学亚型的淋巴瘤其临床病理特点各不相同, MCL常见于50岁以上成人, 好发部位为回肠末段及空肠, 呈多个息肉样改变, 故又称多发性淋巴瘤性息肉病[25,26]. IPSID常见于大龄儿童或青年, 好发于近段小肠, 患者常有腹泻、腹痛表现[27]. Burkitt lymphoma常见于儿童, 与EBV或HIV相关[28,29]. 小肠T细胞来源淋巴瘤以肠病相关T细胞淋巴瘤为主, 易于Crohn's病混淆[30]. 滤泡性淋巴瘤常见于十二指肠[31-33].

2.2 实验室诊断

2.2.1 消化道钡餐: 消化道钡餐检查是消化道病变常规的检查手段, 大部分PSIL病例通过影像检查包括气钡双重对比灌肠造影及CT扫描. 影像学表现可为肠壁局部或全周性增厚、结节或肿物, 邻近肠袢紊乱, 也可有表面溃疡, 溃疡样不规则形态, 坏死性肿块, 局段性肠管的狭窄和僵硬, 其不足是不能定性诊断, 也不能提供是否伴有淋巴结及其他器官累及等方面的信息[34].

2.2.2 内镜及超声: 内镜检查在消化系统疾病中

占有非常重要的地位, 十二指肠起始段肿瘤可以通过胃镜发现, 末端回肠可凭借结肠镜作逆行检查, 胶囊内镜是目前筛选小肠疾病的有效无创手段, 对于小肠炎症, 血管病变, 隆起性病变及息肉均可经胶囊内镜发现, 为小肠疾病进一步诊治提供依据[35-37]. 其阳性率明显高于消化道钡餐造影. 其弊端在所获图片资料不能将PSIL与其他小肠疾病鉴别[38], 且不能取活检, 对肠腔狭窄者有发生梗阻的危险. 新近在临床上应用的小肠镜[39]可对全小肠进行全面的观察, 同时直视下进行活检, 为小肠病变的术前诊断能提供组织学诊断. 肿瘤一般生长于小肠黏膜下, 活检组织要包括黏膜下层组织, 否则易漏诊, 必要时需进行多次活检. 超声检查对于PSIL敏感性差, 其主要用于检查淋巴结及其他器官有无转移.

2.2.3 *多排螺旋CT及PET/CT*: 多排螺旋CT也有助于PSIL的诊断. 其CT征象为肠壁增厚, 肠腔内息肉样肿块, 肠管扩张, 肠套叠等, 增强扫描病灶呈轻至中度强化. 再利用虚拟内镜软件对多排螺旋CT图像进行后期处理, 即多排螺旋CT仿真内镜(multi-detector CT virtual endoscopy MDCT-VE), 有文献对MDCT-VE诊断小肠肿瘤进行分析, 其敏感性达90.9%、特异性98.9%、准确率96.8%[40].

将CT高的解剖学分辨率和PET获取的分子水平的功能与代谢信息相结合的^{18}F-DG PET/CT[41]大大提高了肿瘤的检测率, 被称为“世纪分子”的^{18}F-DG是一种天然的葡萄糖类似物, 是目前应用最为广泛的肿瘤显像剂, 主要反映肿瘤的能量代谢, 其摄取强度与肿瘤的恶性程度和预后相关, 由于淋巴肿瘤组织的代谢增强, 葡萄糖酵解增强, 淋巴瘤细胞摄取大量的^{18}F-FDG, ^{18}F-FDG在磷酸己糖激酶作用下形成FDG-6-磷酸长时间停留在淋巴瘤细胞内, 显示放射性浓聚. 同属无创性检查, ^{18}F-FDG PET/CT在结外淋巴瘤的诊断、分期评价中较CT更敏感[42], 能早期发现淋巴瘤病灶, 敏感性和特异性较高, 有助于临床准确诊断、拟定治疗方案和判断预后[43,44]. 但PET/CT常受淋巴瘤的分化程度、炎性组织及肉芽组织的影响, 存在一定比率的假阳性和假阴性, 尚需寻找一种新的显像剂对其进行甄别和其他影像学手段进行鉴别诊断.

近年来, 也有利用微创腹腔镜检查直接取可疑病灶行病检, 对PSIL的术前评估、术中探查、快速病检均有较大帮助. 总之, PSIL暂无特异性诊断方法, 早期诊断较困难, 确诊必须依赖于内镜下或术后病理活检. CT、PET/CT、超声与内镜检查主要用作非侵袭性方法, 对PSIL分期和患者的随访有重要[45].

3 治疗

PSIL的治疗目前尚无统一的最佳方案, 其临床表现分期的不同, 治疗方法和临床效果也有不同, PSIL的治疗方式包括单纯外科手术、化疗、放射治疗及生物治疗以及综合治疗. 其中外科手术结合化疗是最主要的治疗方式[46,47]. 手术可以明确肿瘤的侵犯范围及病理分类, 有助于下一步治疗的选择, 并且切除肿瘤能减轻放疗、化疗的负荷. 但也有学者认为手术和放疗加化疗(保守治疗) 疗效无差异, 保守治疗能更好地保存小肠的功能. 加拿大玛格丽特女王医院的资料表明, 在近10年内应用放、化疗结合使胃肠淋巴瘤的10年生存率较单一手术或放疗的88.0%, 延长到99.5%[48].

3.1 *手术治疗* 小肠淋巴瘤局部手术可切除肿瘤原发灶及部分可能有转移的淋巴结, 可解除梗阻, 疗穿孔、出血等严重并发症, 甚至为病理分型及临床分期提供直接依据, 同时起到诊断和治疗作用[49,50]. 早期的B细胞来源的PSIL(stage IE)只需行单纯病变肠段切除术. 据区域性小肠淋巴瘤的部位选择不同的手术方式: 十二指肠三、四段淋巴瘤可行肠切除术; 若十二指肠近端或累及乳头或已侵犯胰腺的淋巴瘤, 可行胰十二指肠切除术; 回肠淋巴瘤需行右半结肠切除术[51]. 对有梗阻和穿孔的病变局限的肠段可行手术切除, 并结合全腹的放化疗. 有明显淋巴结肿大且术前已有其他脏器转移者需行广泛淋巴结清扫术, 减少淋巴瘤的负荷, 有利于局部病灶的控制, 减少在辅助治疗过程中, 未切除肿瘤引起的出血与穿孔, 进而导致病情的进一步恶化[52]. 原发性小肠淋巴瘤以弥漫性大B细胞淋巴瘤(DLBCL)最为多见, 高恶度淋巴瘤在小肠占较大比例, 分期和治疗方案与胃淋巴瘤相同, 但肠道晚期病例较多, 手术切除率较低. 单纯依靠手术治疗难以达到临床根治效果, 因而辅助治疗成为PSIL治疗的重要组成部分[53].

3.2 *放疗与化疗* 在手术风险大, 手术不能改善预后者或手术不能完全切除或有区域淋巴结转移情况下, 考虑行单纯放、化疗或辅助放化疗, 可明显延长患者的生存期. 一些文献报道放疗对局部发生的小肠淋巴瘤有较好治疗效果[54], 尤其

■**创新盘点**
对小肠淋巴瘤的诊治方案可以结合分子细胞免疫生物学, 更科学的应用于临床, 提高检出率, 促进其预后.

■应用要点
此文对临床少见疾病原发性小肠淋巴瘤临床病理特点、诊断、治疗及预后进行了综述, 但着重于临床, 强调诊断及治疗, 具有较强的临床指导意义.

是发生于十二指肠[55], 但放疗对小肠的DLBCL作用效果并不佳[56], 可能与其多灶性及弥漫性有关. 恶性淋巴瘤对化疗均敏感, 单纯化疗B淋巴细胞来源较T淋巴细胞来源的小肠淋巴瘤效果好[57,58], 可消灭残留组织肿瘤细胞, 并提高治疗效果. 据文献报道, 手术加化疗治疗原发性胃肠道淋巴瘤(PGML), 5年生存率可达73%, 治疗胃黏膜相关淋巴组织淋巴瘤5年生存率可达90%. Koh等[59]报道胃MALT45例, 手术加化疗或抗HP治疗加化疗, 5年、10年生存率分别为52%、45%.小肠淋巴瘤16例外科手术或加化疗, 5、10年生存率均为60%. Samel等[60]报道66例肠道大细胞性淋巴瘤, 单纯手术占16%, 综合治疗占84%, 5年生存率为58%, 10年生存率为48%, 综合治疗组CR率、中位无瘤生存期优于手术治疗, 但生存率差异无显著意义.

有些学者主张对于早期无症状的FL(stage IE)采用期待疗法, 对于有症状或者晚期的FL则采取手术加化疗或手术加放疗, 化疗一般采用CHOP方案. MCL化疗后只能短时间的缓解, 其对化疗反应和预后极差. 其治疗因患者能否行干细胞移植而异. 对于能接受移植的患者, 在移植前一般采用R-CHOP或R-Hyper CVAD方案治疗, 反之则单独使用Rituximab或者核苷酸类似物联合Rituximab治疗. 早期IPSID利用抗生素如四环素联合甲硝唑、氨苄西林, 6-12 mo可缓解. 中晚期IPSID则需要用蒽环类化疗药联用抗生素治疗[61]. 有报道66%的EATL术后辅助化疗或行自体干细胞移植术可达到持续完全缓解[62].

应注意的是放、化疗容易引起急性反应, 如肠出血、肠穿孔、血管炎、肠炎等[63]. 对长期放化疗的患者有晚期效应的不良反应, 最常见的就是继发恶性肿瘤, 心脑血管疾病, 对于这类患者应密切随访以及早发现并及时干预治疗.

3.3 生物治疗 PSIL大多是B细胞非霍奇金淋巴瘤, 90%以上B细胞非霍奇金淋巴瘤表达CD20抗原, PSIL生物治疗临床常用美罗华(利妥昔单抗, Rituximab), 其定向作用于B细胞表面的CD20抗原. 美罗华联合化疗治疗B细胞型非霍奇金淋巴瘤疗效显著, 使临床缓解率及中位生存率都明显提高, 患者耐受良好, 是当前临床治疗CD20阳性的B细胞性NHL的首选方案[64]. 但其作用效果及对预后的影响仍受争议[65]. 随着人们对单克隆抗体在胃肠道淋巴瘤发病过程中作用的认识, 逐渐研发了第二、三代抗CD20抗体(ofatumumab, veltuzumab,ocrelizumab), 抗CD22抗体Epratuzumab, 抗CD30抗体SGN-30, 抗CD40抗体SGN-40, 抗血管内皮生长因子(VEGF)抗体becacizumab[66], 抗CD52抗体阿仑珠单抗(alemtuzumab)治疗T细胞型原发性小肠非霍奇金淋巴瘤也取得一定的疗效[67].

3.4 其他 其他治疗方案如造血干细胞移植、免疫增强剂、PET/CT、生物标记、中医中药等治疗均有报道[68].由于大量免疫抑制剂的运用等多种因素影响, PSIL发病率有升高的趋势, 所以小肠淋巴瘤的诊断及治疗还需进一步探究和改进.

4 结论

影响PISL预后因素有: 肿瘤浸润范围, 临床分期, 组织学类型, 免疫表型, 有无并发症及严重程度[69,70]. 目前国际预后指数(IPI)对几乎所有的非霍奇金淋巴瘤有重要参考价值. 近年来, 小肠疾病的辅助检查方法的不断改进及新的检查方法的出现, PSIL更容易被早期诊断, 继而积极采取有效的治疗手段, 大大促进了PSIL的预后的改善. 随着对恶性淋巴瘤的分子生物学、分子免疫学研究如分子免疫表型研究、单克隆抗体靶向治疗等都取得了一定进展, 这对恶性淋巴瘤的诊断、治疗方法的改进都起了一定的促进作用, 但仍有不少问题需要深入研究.

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■同行评价

本文综述了原发性小肠淋巴瘤的临床病理特征及诊治进展, 综述文献合理、全面, 立题新颖, 文章侧重于临床, 有利于对小肠淋巴瘤的临床表现的进一步认识, 结合临床实践, 对临床诊治有指导意义.

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