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胃肠间质瘤诊疗进展

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摘　要

胃肠间质瘤(GIST)诊断需结合临床表现(如胃肠道出血、腹痛等)、增强CT、超声内镜、MRI、影像组学等影像学检查以及CD117、CD34等免疫组化指标。治疗以手术治疗为核心，包括开腹、腹腔镜、内镜治疗及双镜联合、达芬奇辅助手术等；药物治疗以伊马替尼等酪氨酸激酶抑制剂(TKI)为主，涵盖术前新辅助、术后辅助及转移复发/不可切除病例的阶梯治疗，免疫靶向治疗等。随着分子生物学的发展，对GIST的理解日益深入，GIST的多种诊断技术和治疗都有了新的进展，特别是在个体化、精准医疗和靶向治疗方面有极大的进步。

关键词

胃肠道间质瘤，增强CT，超声内镜，影像组学

Advances in the Diagnosis and Treatment of Gastrointestinal Stromal Tumors

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Abstract

The diagnosis of gastrointestinal stromal tumor (GIST) requires a combination of clinical manifestations (such as gastrointestinal bleeding, abdominal pain, etc.), imaging examinations including contrast-enhanced computed tomography (CT), endoscopic ultrasonography (EUS), magnetic resonance imaging (MRI) and radiomics, as well as immunohistochemical markers such as CD117 and

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CD34. Surgery serves as the core of treatment, including open surgery, laparoscopic surgery, endoscopic therapy, combined dual-endoscopy surgery and da Vinci robot-assisted surgery. Pharmacotherapy is mainly based on tyrosine kinase inhibitors (TKIs) such as imatinib, covering preoperative neoadjuvant therapy, postoperative adjuvant therapy, stepwise therapy for metastatic/recurrent or unresectable cases, and immune-targeted therapy. With the development of molecular biology, the understanding of GIST has been increasingly in-depth, and various diagnostic techniques and treatments for GIST have achieved new progress, especially great advances in individualized and precision medicine as well as targeted therapy.

Keywords

Gastrointestinal Stromal Tumor, Contrast-Enhanced CT, Endoscopic Ultrasonography, Radiomics

1. 引言

胃肠间质瘤(GIST)起源于胃肠道 Cajal 间质细胞，作为消化道最常见的间叶源性肿瘤，GIST 可发生于整个消化道，原发肿瘤位置以胃部位最为常见(55.6%)，其次是小肠(31.8%)、结直肠(6.0%)、其他/多部位(5.5%)和食管(0.7%) [1] 。其全球发病率为(0.4~15.6)/10 万人，我国每年发病率约 0.79~1.02 例/10 万人。GIST 具有不同程度的恶性潜能[2]，其侵袭性与其临床表现及后续治疗和预后密切相关，解剖位置、肿瘤大小和有丝分裂率是预测病理危险度的重要因素[3]。GIST 已进入精准医疗时代，随着对 GIST 生物学行为认识的不断加深，以及诊疗技术的不断进步，在早期准确诊断和治疗上取得了很多关键性的进展。本文结合目前研究成果与临床实践系统阐述 GIST 诊疗的现状，现报道如下。

2. 术前诊断

2.1. 临床表现

无特定患病年龄群，多数患者集中在 50~80 岁[4]。GIST 可发生于消化道的任何部位，其临床症状无特异性，与发生的部位、大小及生长方式相关[5]，常见症状包括胃肠道出血、腹痛[6]，部分存在体重减轻、腹胀或腹部肿块[4]，严重时可出现肿瘤破裂或胃肠道梗阻[7]。

2.2. 影像学检查

2.2.1. 增强 CT

辨别 GIST 的首选影像方法[8]，增强 CT 可清晰显示肿瘤的大小、形态、内部密度、周围组织的解剖关系及对于肿瘤的强化方式[9]，其影像学特征与病理危险等级密切相关。Li 及其同事构建了术前预测 gGIST 恶性潜能的 CT 列线图(nomogram)模型，实现了对原发性 gGIST 恶性程度的术前精准预测与风险分层[10]；Jung 等人[11]的研究表明 CT 可预测 GIST 治愈切除后复发；Chen [12]等人建立了基于 CT 特征的术后无病生存期(DFS)预后评估模型，阐述了 CT 对术后复发风险的精准评估与分层。增强 CT 既为 GIST 的早期诊断、恶性潜能预判提供可靠依据，也为治疗方案制定、术后随访及预后评估提供全方位支撑，在 GIST 诊疗全流程中发挥着不可替代的关键作用。基于增强 CT 技术衍生出的能谱 CT [13]可分析不同能量水平下的物质吸收特性，能够进行组织成分定量分析，在 GIST 的早期检出、风险分层、治疗反

应评估等方面展现出显著优势。陈楠等人[14]发现能谱 CT 成像通过多参数应用对胃黏膜下微小病变的检出率有较增强 CT 有显著的提升。Grazzini 等人[15]表明能谱 CT 可以准确预测原发性胃肠道间质瘤的恶性潜力。Meyer 等人[16]开展前瞻性研究，建立了接受 TKI 治疗的 GIST 患者的疗效反应参数评估体系，实现了对 TKI 治疗效果及患者预后的精准预测。

2.2.2. 超声内镜(EUS)

EUS 可提供基于传统白光内镜的病变内部超声、起源、异质性、生长模式、胃肠壁内的侵入情况及与血管关系等信息。GIST 的表现为低回声、不均质、无回声或高回声(肿瘤为恶性时)，通常位于第三和第四层，极少数位于第二层[17]。EUS 在 GIST 的诊断、风险评估中具有重要意义，在区分黏膜下肿瘤与外源压迫方面具有极高的敏感性和特异性。Chen [18]及 Liu [19]认为 EUS 可以对 GIST 的恶性潜能进行风险预判；Mi 等[20]人认为 EUS 在术前诊断、手术方案规划及术中引导中起关键作用。Sekine [21]等研究阐明 EUS-FNA 技术对 GIST 的精准定性诊断及区分病变性质的特点。

2.2.3. MRI

能够提供肿瘤大小、穿孔、转移及肿瘤侵入邻近结构的信息，具有多参数和高软组织分辨率的优势。Lanke [22]等认为其在识别肝转移、出血和肿瘤坏死时比 CT 更准确；Yoo [23]认为 MRI 对 GIST 与良性胃上皮下病变的鉴别诊断有很大帮助，实现了对两类病变的精准区分。可用于评估 GIST 风险，Zheng 等[24]学者通过 MRI 分析技术建立模型明确了对 GIST 风险的精准分层。

2.2.4. PET-CT

基于组织代谢的变化，使用 18F-氟二氧葡萄糖(FDG)检测癌症，对判断肿瘤恶性程度、预后评估及治疗后反应效果有一定参考价值。Li 等[25]研究者建立了基于(18F-FDG)PET/CT 的 gGIST 良恶性鉴别诊断体系实现了对原发性 gGIST 良恶性的精准区分，Du 等学者也同意此观点[26]。可用于预后风险预测和监测疾病进展，Albano 等人[27]分析了治疗前 gGIST 的代谢行为，建立了预后评估模型，实现了对 gGIST 患者预后风险的精准分层。Dimitrakopoulou 等[28]建成多模态影像技术，用于评估早期 TKI 治疗反应。

2.2.5. 影像组学

影像组学为 GIST 的定量分析与个体化评估提供了新途径，其核心是从 CT、MRI、内镜超声(EUS)等影像中提取纹理特征、灰度直方图特征、小波变换特征等肉眼无法识别的高通量特征，进而构建分析模型。目前已有多项研究验证了影像组学在 GIST 诊疗全流程中的应用价值：Zhu 等[29]开发了 MMP-AI 的系统通过 WLE 或 EUS 图像识别突出的胃部病变；Jia 等[30]构建了 CT 放射学模型用于 1~2 cm gGIST 的术前 GIST 危险度分级；Meyer 等[16]设计一项前瞻性、多中心研究，采用 DECT ViTB 评估接受 TKI 治疗的 GIST 患者的治疗反应。Ji 等[31]根据临床数据和术前 Delta-CT 影像学建模，预测 GIST 的复发情况。影像组学能够覆盖 GIST 从无创诊断、危险度分层、疗效评估到预后预测的全流程精准管理[32]，为 GIST 的个体化诊疗提供了重要工具。

2.3. 液体活检

是一种简单且非侵入性的癌症生物标志物检测方法，最常见的是循环肿瘤 DNA (ctDNA)，循环肿瘤 DNA (ctDNA)指的是识别癌细胞排出到血液中的 DNA 片段，在 GIST 识别新颖 KIT 突变、检测肿瘤进展、药物疗效反应及预后方面具有切实价值。Bleckman 研究[33]发现肿瘤大小与 GIST 患者 ctDNA 检测之间存在相关性，肿瘤越大越可能脱落可检测的 ctDNA。Rassner 等[34]学者建立高灵敏度数字液滴 PCR 用于检测 ctDNA 以实现耐药突变的精准检出；Serrano 等[35]聚焦晚期 GIST 患者 KIT 突变的分布特点，

揭示了 ctDNA 可作为评估患者靶向治疗疗效、预测预后的潜在分子标志物。但存在一定局限性，但 Brinch 等人[36]发现极低至中等风险 GIST 患者几乎不分泌或极少 CTNA，ctDNA 在高风险 GIST 患者中的相关性尚不明，ctDNA 的碎片化特性使得检测复杂耐药突变具有挑战性。

2.4. 病理诊断

2.4.1. 病理学特征

GIST 多为分布良好的单发病变，大小不一，其肉眼呈灰白色，常伴出血灶、中心囊性变性或坏死[37]。肿瘤细胞形态可分为有嗜酸性纤维质的纺锤体细胞型(70%)、具有透明嗜酸性细胞质的上皮型(20%)，以及具有纺锤体和上皮细胞的混合型(10%) [38] [39]。危险度分级以中国 GIST 专家共识 2017 版在 NIH (2008 版)基础上进行优化的改良版(表 1)为标准，依据肿瘤大小和核分裂象计数，极低风险 GIST 发生率最低(15%)，低、中、高风险类别分别占 30%、22%和 33% [1]。

Table 1. Risk stratification of primary gastrointestinal stromal tumor (modified NIH 2008 criteria)
表 1. 原发胃肠间质瘤危险度分级(NIH 2008 改良版)

危险度分级	肿瘤大小	核分裂象/50HPF^{-1}	肿瘤原发部位
极低	≤2	≤5	任何
低	2.1~5.0	≤5	任何
中等	2.1~5.0	6~10	胃
	≤2	6~10	任何
	5.1~10.0	≤5	胃
高	任何	任何	肿瘤破裂
	>10	任何	任何
	任何	>10	任何
	>5	>5	任何
	>2，≤5	>5	非胃原发
	>5，≤10	≤5	非胃原发

2.4.2. 免疫组织化学评估

GIST 的主要发病机制与 KIT 或 PDGFRA 基因的特异性突变密切相关[40]。GIST 中 CD34 在 GIST 中表达特异性及阳性率约达 70% [4]；CD117 在约 95%的 GIST 中表达，其与 CD34 联合应用可提高诊断准确性。GIST 中有 95%的患者表达 DOG1 [38]，但是大多数非 GIST 肿瘤 DOG1 也表现为阳性，故不作为特异性标志物。α-SMA 是肌动蛋白的聚合物形成肌动蛋白丝，许多 GISTs (以小肠 GISTs 为著)可检测出该标志物[41]。Ki-67 指数与肿瘤复发及无进展生存期相关，可辅助预后评估。

3. 治疗

3.1. 手术治疗

2025 年 CSCO 胃肠间质瘤诊疗指南明确指出：GIST 手术治疗基本原则是保证切缘的组织学阴性，不推荐常规行淋巴结清扫术，术中避免肿瘤破裂并注意保护肿瘤假性包膜完整，手术目标是尽量争取 R0 切除。对于直径 ≤2 cm 的胃 GIST 伴临床症状者考虑行手术切除，无症状患者根据其内镜和 EUS 表现确定是否具有进展风险并定期随访。直径 >2 cm 的 GIST 评估无手术禁忌证手术切除是首选的治疗方法。

3.1.1. 开腹手术

传统手术方式，适用于肿瘤较大、侵犯范围广或合并复杂情况的 GIST，能确保完整切除病灶，但创伤相对较大。

3.1.2. 腹腔镜手术

微创手术的发展使 GIST 尤为适合腹腔镜切除[42]，已报道采用多种手术方法，如腹腔镜楔形切除、外翻切除和胃部次全切除等手术方法。腹腔镜手术被视为肿瘤直径为 2~5 cm GIST [43]的标准手术，同时也适用于 5 厘米以上的 GIST，Lian 等[44]的研究支持这一观点。Goh [45]、MacArthur [46]、Chen [47]及 Schmidt [48]等研究显示腹腔镜手术较传统开腹对 GISTs 的侵入性更小、疼痛更小、并发症更少、耗时短，术后恢复更快，美容效果也优于开腹手术，且肿瘤预后相似。

3.1.3. 内镜治疗

治疗方式包括内镜黏膜下挖除术、黏膜下隧道内镜切除(STER)、内镜全层切除(EFTR) [49]等，根据 2020 年 GIST 内镜指南适用于直径 ≤2 cm 的有症状 GIST 或 2~5 cm 的低风险 GIST。Liu 等[50]使用荟萃分析充分证明了内镜治疗是针对 gGIST 小于 2 厘米和 2~5 厘米范围内的腹腔镜手术治疗的一种安全高效的替代手术，且不会恶化肿瘤结局。Zhang 等[51]认为对于直径 < 4 cm 的 GIST 有较好的疗效，Zhu 等[52]分析比较内镜治疗与腹腔镜切除术两种治疗手段，明确内镜治疗有最大程度保留消化道生理结构，住院时间短、术后并发症少等优点。

3.1.4. 双镜联合技术

对于特殊位置(如胃后壁、食管胃结合部)、特殊生长方式(如跨壁生长、腔内－腔外混合生长)的 GIST，从外部进行外科手术较为困难，故可结合内镜与腹腔镜的优势[53]，尽量减少切除面积及保留生理功能。Hiki [54]及 Hashimoto [55]等学者明确了该术式在 GIST 治疗中具有微创、精准、器官功能保留好的优势，且安全性与有效性得到多中心验证。Namikawa [56]认为使用该法可应用于<5 cm GIST，其优点是切除面积减少、失血量更低。

3.1.5. 达芬奇辅助手术

作为微创外科的先进技术，其安全性得到肯定，可提供更清晰的视野和灵活的操作，适用于复杂 GIST 的切除，有助于减少手术创伤、缩短恢复时间。Ceccarelli [57]及 Jian [58]等人研究表明机器人手术对于直径 > 5 cm、在复杂部位的 GIST 在精细操作、消化道重建等方面更有优势。

3.2. 药物治疗

3.2.1. 术前药物治疗(新辅助治疗)

70%~80%的 GIST 携带 KIT 原癌基因突变故伊马替尼对 GIST 患者有明确疗效。NCCN 指南[59]建议在手术前使用新辅助伊马替尼治疗，考虑用于肿瘤较大且无法立即切除的患者，用以减小肿瘤大小，并降低原发性 GIST 患者被认为不可切除或可切除但高风险发病率的发生风险，Jakob [60]及 Joseph [61]等的研究证明此观点。

3.2.2. 术后辅助治疗

中国 2024 年 GIST 诊疗指南建议中高危患者行治愈性手术后推荐伊马替尼术后辅助治疗，Joensuu [62]的研究同指南观点相同。DeMatteo 等[63]人研究认同术后 1 年佐用伊马替尼甲已被证明能延长整体存活期；Joensuu [64]明确 3 年伊马替尼辅助治疗能显著延长患者无病生存期，尤其对携带 KIT 外显子 11 突变的患者获益更显著；Raut 等[65]学者聚焦 5 年伊马替尼辅助治疗能显著延长患者无病生存期，且安全

性良好，目前术后治疗的最佳持续时间尚不明确。

3.2.3. 转移复发/不可切除 GIST 的治疗

指南建议依序使用酪氨酸激酶抑制剂(TKI)伊马替尼、舒尼替尼、瑞可拉非尼和利普替尼治疗[66]，这些药物用于治疗不可切除或转移性(U/M) GIST，分别用于第一、二线、第三线和第四线治疗[67]。GRID试验[68]证实雷戈拉芬尼在伊马替尼和舒替尼难治性 GIS 患者中显著延长了无进展生存期的持续时间，已成为晚期 GIST 患者的重要治疗选择，于 2013 年成为标准的三线治疗。利普替尼是一种新型“开关控制”TKI，旨在抑制广泛谱系的 KIT 和 PDGFRA 突变，从而减轻驱动肿瘤生长的异常信号[69]，INVICTUS试验[70]显示利丙替尼显著延长了中位无进展生存期(安慰剂组为 6.3 个月，安慰剂组为 1.0 个月)。D842V突变使激酶对 TKI 有较大耐药性，阿伐普替尼是首个对 PDGFRA D842V 突变表现出临床显著活性的TKI，缓解率高达 88% [71]，且能显著降低疾病进展风险、延长患者生存，同时验证了其安全性可控。

3.2.4. 免疫治疗及新治疗

免疫检查点抑制剂：单独使用效果有限，与伊马替尼联合使用可增强抗肿瘤作用，相关联合治疗研究正在进行。

其他潜在治疗：细胞因子治疗、抗 KIT 单克隆抗体、PD-1 和 PD-L1 抗体、CTLA4 单克隆抗体、细胞治疗、HSP90 抑制剂[72]、选择性核输出抑制剂、DS-6157a [73]等均处于研究阶段。

4. 展望

尽管 GIST 诊疗取得了显著进展，但仍面临诸多挑战，未来 GIST 诊疗将聚焦三大方向：一是完善多组学整合诊断体系，优化影像组学与基因组学的联合模型，提高危险度分级与疗效预测的精准性；二是深入研发针对突变的新型靶向药物，优化治疗方案，明确术后治疗的最佳持续时间，拓展新型治疗手段的应用；三是深化人工智能技术融合，推动 AI 辅助手术规划、实时疗效监测等功能的临床转化，实现真正意义上的个体化精准医疗。随着这些领域的持续突破，GIST 患者的生存质量与远期预后将得到进一步提升。

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