

胃神经鞘瘤伴淋巴结肿大及溃疡形成一例并文献复习

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收稿日期: 2026年4月9日; 录用日期: 2026年5月2日; 发布日期: 2026年5月12日

摘要

胃神经鞘瘤(gastric schwannoma, GS)是起源于胃黏膜下神经丛施万细胞的间叶性肿瘤, 由于缺乏特异临床表现, 常被误诊为胃肠间质瘤或胃癌。本文报告四川省人民医院诊治的1例术前影像学及相关检查高度提示恶性, 但术后免疫组化证实为胃神经鞘瘤的患者, 术后长期随访未见复发。结合相关文献对其临床特点及诊断要点进行分析, 以期提高对该病的认识。

关键词

胃神经鞘瘤, 淋巴结肿大, 溃疡, 胃癌, 免疫组化

Gastric Schwannoma with Lymphadenopathy and Ulceration: A Case Report and Literature Review

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Received: April 9, 2026; accepted: May 2, 2026; published: May 12, 2026

Abstract

Gastric schwannoma (GS) is a mesenchymal tumor arising from Schwann cells of the gastric

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文章引用: 沈光鹏, 谢晨飞, 杨洲. 胃神经鞘瘤伴淋巴结肿大及溃疡形成一例并文献复习[J]. 临床医学进展, 2026, 16(5): 682-687. DOI: 10.12677/acm.2026.1651861

submucosal nerve plexus. Due to the lack of specific clinical manifestations, it is often misdiagnosed as gastrointestinal stromal tumor or gastric cancer. This article reports a case from Sichuan Provincial People's Hospital, where preoperative imaging and related examinations highly suggested malignancy, but postoperative immunohistochemistry confirmed the diagnosis as gastric schwannoma. No recurrence was observed during long-term postoperative follow-up. By reviewing relevant literature, we summarize the clinical characteristics and key diagnostic points of GS to improve clinicians' recognition of this disease.

Keywords

Gastric Schwannoma, Lymphadenopathy, Ulcer, Gastric Cancer, Immunohistochemistry

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1. 病历资料

患者，男性，41岁，因左侧腰背部疼痛1周于2023年11月入院，疼痛呈间歇性隐痛，与进食无明显关系，无黑便、腹胀、恶心、呕吐等症状，既往10余年前因胆囊结石行腹腔镜胆囊切除术，入院查体未见明显异常。

影像学检查：外院增强CT提示胃小弯约60 mm × 50 mm软组织肿块，伴周围多发淋巴结肿大。胃镜示胃体小弯侧见一大约5 cm黏膜下病变，表面可见溃疡形成。我院增强CT显示病灶侵及浆膜层，贲门旁、胃左动脉旁及腹腔干走行区可见多发肿大淋巴结(见图1)，肝左外叶见直径约20 mm稍低密度结节，考虑肝血管瘤，影像学考虑胃癌伴淋巴结转移(T4aN2Mx)。超声内镜(EUS)提示胃小弯见一巨大溃疡型低回声肿物(约45 mm × 46 mm)，边界欠清，并见腹腔干旁多发低回声淋巴结(见图2)，肿物性质不明，考虑分期为uT4N1M0。

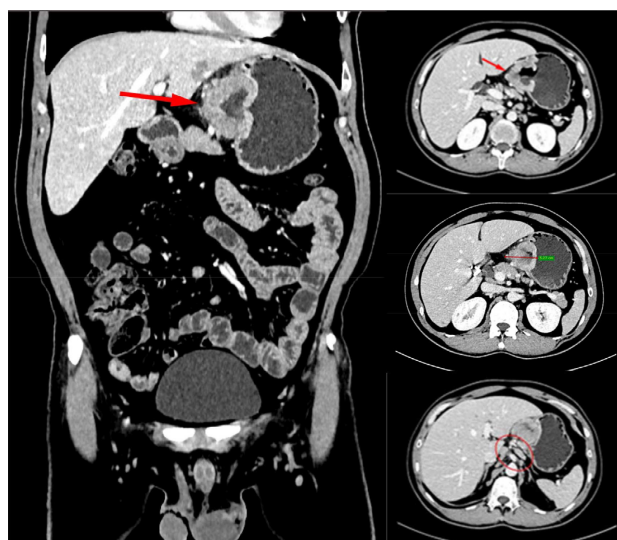


Figure 1. Contrast-Enhanced abdominal CT

图1. 腹部增强CT图像

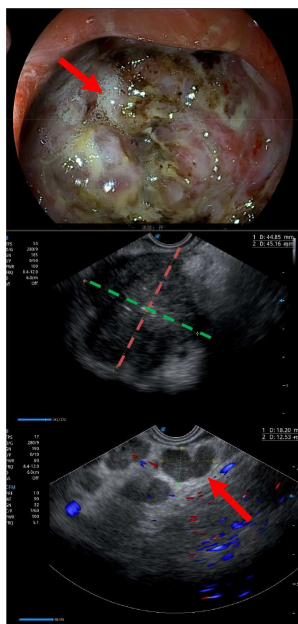


Figure 2. Endoscopic ultrasonography (EUS)
图 2. 超声内镜检查图像(EUS)

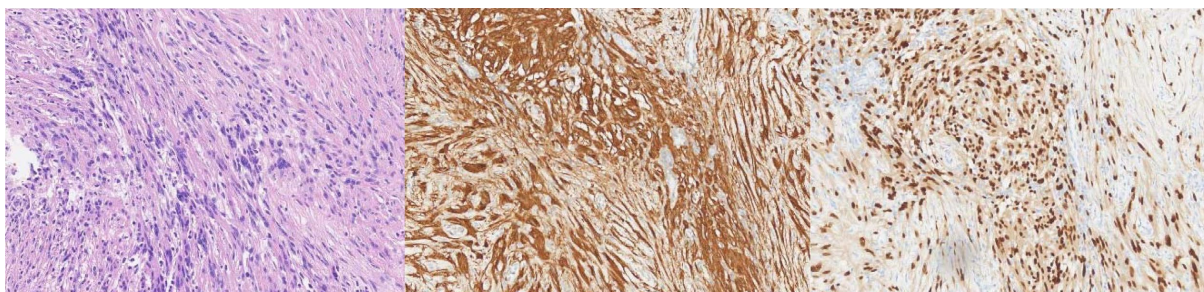


Figure 3. Pathological with immunohistochemical staining images
图 3. 病理切片及免疫组化图像

术前实验室检查示血清铁蛋白 446.23 ng/mL、癌胚抗原 7.89 ng/mL、超敏 C 反应蛋白 33.46 mg/L 及白细胞 $9.78 \times 10^9/L$ ，均轻度升高。EUS 引导下细针穿刺病理检查示胃体组织少量梭形细胞，不能排除胃肠间质瘤，淋巴结穿刺细胞学提示“退变核异型小细胞团，不能排除转移性恶性肿瘤”，但液基制片同时报告未见肿瘤细胞，结果存在不一致。外院活检及 EUS-FNA 标本行免疫组化均显示 S100(+)、SDHB(+)、CD117(-)、DOG1(-)，提示胃肠型神经鞘瘤可能，建议完整切除后进一步确诊。

2. 治疗与随访

术前综合评估高度提示恶性，遂于 2023 年 12 月 4 日行远端胃切除术 + 淋巴结清扫 + Roux-en-Y 吻合，术中见胃小弯肿瘤约 5 cm，肿瘤侵及肌层，表面可见溃疡，第 5 组淋巴结肿大并部分融合。术后病理示梭形细胞增生，免疫组化显示 S100(+)、SOX10(+)、SDHB(+)、CD117(-)、DOG1(-) (见图 3)，Ki-67 约 8%，确诊为胃神经鞘瘤。共清扫淋巴结 19 枚，均为反应性增生(0/19)。术后随访 27 个月，患者 CEA 呈轻度波动性升高(5.71~14.16 ng/mL)，但同期影像学及内镜检查均未见肿瘤复发征象，胃镜检查仅提示残胃炎，吻合口及残胃未见新生物。

3. 讨论

胃神经鞘瘤是起源于施万细胞的罕见间叶性肿瘤,多为良性[1]-[3],常表现为边界清晰的黏膜下肿块,但部分病例可伴明显的肿瘤周围淋巴结反应性增生[4] [5],由于位于黏膜下层,常规内镜活检取材有限,术前诊断较为困难[6],EUS-FNA 虽可获取深部组织,但仍可能因肿瘤细胞稀少及炎性细胞混杂而影响判断。本病例术前多项证据高度提示恶性:CT 及胃镜等影像学检查均见病灶表面溃疡形成、黏膜中断并累及肌层,伴多发淋巴结明显肿大,肿瘤标志物及炎症指标升高;术前淋巴结细胞学检查结果前后不一致,进一步增加了诊断难度。

术后病理证实 19 枚淋巴结均为反应性增生,未见肿瘤转移,这与文献报道胃神经鞘瘤典型的瘤周淋巴细胞套(peritumoral lymphoid cuff)表现一致[4]。这一现象可能源于肿瘤相关免疫反应与局部炎症的协同作用:一方面,胃神经鞘瘤细胞高表达施万细胞相关抗原(如 S100、SOX10),可激活适应性免疫应答,促进淋巴细胞在淋巴结中的募集与扩增;另一方面,肿瘤表面伴溃疡形成后,坏死组织释放损伤相关分子模式(damage-associated molecular patterns, DAMPs),激活机体固有免疫,促进炎性细胞因子(如 IL-6、TNF- α 、IL-1 β)的分泌,增强局部炎症反应。本例患者术前 hs-CRP 为 33.46 mg/L,白细胞计数 $9.78 \times 10^9/L$,血小板计数 $397 \times 10^9/L$,均提示全身炎症状态,为上述机制提供了临床佐证。在这些背景下,区域淋巴结在影像学上表现出难以与转移鉴别的肿大,而淋巴结穿刺细胞学中出现的退变性核异型细胞,更可能是反应性淋巴结中免疫母细胞或组织细胞产生的形态学干扰[7],而非转移性肿瘤细胞。

免疫组化在鉴别诊断中具有决定性作用,胃神经鞘瘤典型表现为 S100(+)、SOX10(+)、CD117(-)、DOG1(-),可与胃肠间质瘤鉴别[8] [9]。本例免疫组化结果符合上述特征,证实为胃神经鞘瘤,病理提示 Ki-67 指数约 8%,略高于文献报道的良性神经鞘瘤水平(多在 0~6%) [10] [11],但术后随访 27 个月未见复发或转移,提示 Ki-67 轻度增高仍可见于良性神经鞘瘤,本例 Ki-67 轻度升高可能与炎性微环境密切相关:一是肿瘤伴溃疡形成,溃疡边缘修复性增生活跃区域的细胞增殖指数本身即可偏高[12],与肿瘤恶性潜能无直接关联;二是肿瘤间质中大量浸润的淋巴细胞、浆细胞及组织细胞均可表达 Ki-67,在混合计数时产生叠加效应[13];三是术前全身炎症状态所伴随的细胞因子环境可能通过旁分泌途径轻度上调肿瘤及间质细胞的增殖相关基因表达。基于上述分析,Ki-67 轻度升高不宜作为判断胃神经鞘瘤恶性潜能或扩大手术范围的独立依据,需结合核分裂象计数、细胞异型性及整体临床病理特征综合判读。

治疗方面,胃神经鞘瘤多为良性,完整切除即可获得良好预后[14]。若术前已明确诊断,可考虑局部切除或腹腔镜肿瘤剜除术以减少创伤[15]。本病例因术前影像学、细胞学均不能排除恶性,且术中见肿瘤侵及肌层、淋巴结肿大融合,故行远端胃切除及淋巴结清扫是符合临床安全原则的治疗策略,对临床有一定警示意义。胃神经鞘瘤复发极为罕见,本病例术后 27 个月未见临床可评估的复发或转移,与文献报道一致[16] [17]。值得关注的是,本病例 CEA 在肿瘤完整切除后非但未持续回落至正常,反而呈波动性升高,术前 CEA 为 7.89 ng/mL,术后 6 个月降至 5.71 ng/mL,此后逐渐上升,术后约 15 个月升至 11.98 ng/mL,术后 27 个月进一步升至 14.16 ng/mL;而同期的影像学及内镜检查均未见肿瘤复发证据,胃镜仅提示残胃炎,吻合口及残胃均未见新生物。术前 CEA 升高更可能与肿瘤表面溃疡所致的炎症反应及黏膜屏障破坏有关[18],同期 hs-CRP (33.46 mg/L)与铁蛋白(446.23 ng/mL)的同步升高可佐证这一机制;术后 CEA 持续波动升高,则更可能由残胃慢性炎症主导,Roux-en-Y 重建后残胃的炎症状态及随访影像所示局部淋巴结轻度变化,均提示局部炎症活动持续存在。综合判断,本例 CEA 升高更可能反映良性炎症过程,而非肿瘤复发信号。所以在胃神经鞘瘤术后随访中,对于孤立性 CEA 轻度升高,应结合内镜及影像学进行动态评估,避免基于单一肿瘤标志物做出过度干预。

4. 结论

胃神经鞘瘤可伴有溃疡形成、淋巴结肿大、肿瘤标志物及炎症指标升高等多种类似恶性肿瘤的非特异性表现，部分病例甚至可能出现细胞学检查异常，增加术前诊断难度。本病例完整展示了从术前高度怀疑恶性到术后确诊为良性神经鞘瘤的诊疗全过程，提示临床在诊治胃黏膜下肿瘤时，即使影像学和细胞学提示恶性可能，也应将胃神经鞘瘤纳入鉴别诊断，并结合免疫组化综合诊断，避免不必要的扩大手术或过度治疗。

声明

该病例报道已获得病人的知情同意。

基金项目

四川省科技厅重点研发项目：2022YFS0223。

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