

沟槽状胰腺炎1例并文献复习

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摘要

沟槽状胰腺炎(groove pancreatitis, GP)是一种少见的局灶性慢性胰腺炎类型,常累及胰腺头部、十二指肠和胆总管之间的区域。因与胰腺癌在临床表现、影像学表现及病理学特征上极其相似,易造成误诊。现报道暨南大学附属第一医院1例沟槽状胰腺炎并行文献归纳,总结对该病的诊治体会。

关键词

沟槽状胰腺炎, 十二指肠旁胰腺炎, 鉴别诊断, 文献复习

A Case of Groove Pancreatitis and Literature Review

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Abstract

Groove pancreatitis (GP) is a rare type of focal chronic pancreatitis, often involving the area between the head of the pancreas, the duodenum, and the common bile duct. Due to its extremely similar clinical manifestations, imaging findings, and pathological features to pancreatic cancer, it is prone to misdiagnosis. This report presents a case of groove pancreatitis at the First Affiliated Hospital of Jinan University, along with a literature review, summarizing the experiences in diagnosing and treating the disease.

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Keywords

Groove Pancreatitis, Paraduodenal Pancreatitis, Differential Diagnosis, Literature Review

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1. 引言

沟槽状胰腺炎(groove pancreatitis, GP)是一种节段性慢性胰腺炎, 该病较为罕见, 多见于国外报道, 国内报道相对较少, 可能与其发病率较低有关[1], 常累及胰头、十二指肠和胆总管之间的解剖区域, 好发于 40~50 岁男性, 常有酗酒史, 临床症状为体重减轻、上腹痛、餐后呕吐和十二指肠狭窄引起的恶心。由于症状缺乏特异性, 易误诊为胰腺癌, 现报道我院一例沟槽状胰腺炎并复习相关文献以提高临床医师对该病的认识及诊治水平。

2. 病例资料



Figure 1. Enhanced CT of the whole abdomen: abnormal density shadow in the uncinate process of the pancreas

图 1. 全腹部增强 CT: 胰腺钩突部异常密度影

患者, 男, 34 岁, 因“腹痛 1 年, 再发加重 3 天”于 2021 年 9 月入院。1 年前患者出现饮酒后上腹部胀痛不适, 曾多次当地医院按“胰腺炎”予以补液对症治疗(具体药物不详)。3 天前患者饮酒后腹痛再发加重, 以上腹部为主, 程度较前剧烈, 伴恶心, 无发热, 无气促, 近期体重下降约 5 kg。既往有高血脂病史 2 年, 吸烟史 16 年, 约 20 支/d, 无饮酒史。体检: 体温(T) 36.5℃, 呼吸(R) 16 次/min, 脉搏(P) 92 次/min, 血压(Bp) 112/76 mmHg。神清, 全身皮肤粘膜无黄染, 双肺呼吸音清, 未闻及明显干湿性啰音。心率 92 次/min, 心律齐, 各瓣膜区未闻及异常杂音。腹平坦, 腹部柔软, 全腹轻压痛, 无反跳痛, 肝脾肾肋下未触及, 无叩痛, 肠鸣音正常。双下肢无水肿。实验室检查: 血常规示白细胞 $9.57 \times 10^9/L$ 、中性粒细胞 60%、红细胞 $4.9 \times 10^9/L$ 、血红蛋白 147 g/L、血小板 $248 \times 10^9/L$ 、超敏 C 反应蛋白 32.36 mg/L; 谷丙转氨酶 14 U/L、谷草转氨酶 15 U/L、总胆红素 10.8 $\mu\text{mol/L}$ 、直接胆红素 2.7 $\mu\text{mol/L}$ 、间接胆红素 8.1 $\mu\text{mol/L}$ 、白蛋白 44.1 g/L; 血淀粉酶 43 U/L; 高敏肌钙蛋白 I 0.007 ng/ml; CA199 9.75 U/ml、CEA 1.55 ng/ml。影像学检查: 全腹增强 CT 示胰头稍肿大, 胰腺钩突部见斑片状弱强化区, 范围约 $1.8 \times 1.6 \text{ cm}$, 周围脂肪间隙模糊见少许渗出, 与邻近十二指肠分界欠清, 十二指肠水平段管壁增厚模糊(见图 1)。全腹 MR 示

胰头周围炎, 胰头体积增大, 周围脂肪间隙模糊, 胰腺勾突周围液化。胃镜见十二指肠球部黏膜鲜红充血水肿, 球腔无变形, 十二指肠降段见一肿物, 表面粘膜充血水肿。局部呈白色颗粒样改变, 环腔半周, 基底暴露欠佳, 质脆, 予取病理送活检(见图 2)。十二指肠活检见(降段)粘膜慢性活动性炎症(见图 3)。超声内镜见胰头增大, 回声减低。考虑胰腺炎可能性大。综合病史体征及辅助检查诊断为沟槽状胰腺炎, 予以禁食、抑制胰酶分泌、抑酸、补液等治疗后患者症状缓解后出院。



Figure 2. Gastroscope: duodenal descending period of the mass in dispute

图 2. 胃镜: 十二指肠降段肿物待查

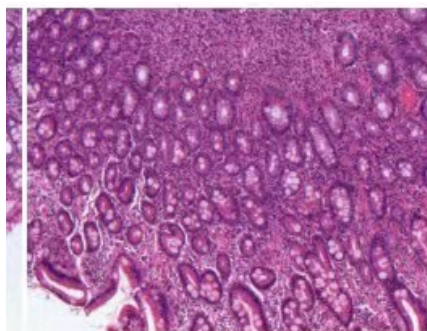


Figure 3. Gastroscope pathological: (drop) mucosal inflammation of chronic activity ($\times 20$)

图 3. 胃镜病理: (降段)粘膜慢性活动性炎($\times 20$)

3. 讨论

沟槽型胰腺炎(groove pancreatitis)是一种局灶性慢性胰腺炎, 累及胰头、十二指肠和胆总管之间的解剖区域。该病首先在 1973 年由 Becker [2]描述, Stolte [3]等人在 1982 年将其翻译为沟槽型胰腺炎, 并分为单纯型(仅侵犯胰十二指肠沟)和节段型(侵犯沟区和胰头)。此外, 许多不同的术语被用于描述沟槽区域的慢性炎症改变, 如十二指肠旁胰腺炎、异位胰腺囊性营养不良、肌腺瘤病、十二指肠壶腹周围壁囊肿和十二指肠胰腺错构瘤[4]-[6], 上述名称都反映了在某些情况下某种因素可能更加突出。

GP 的发病机制尚不十分明确, 一种可能机制为多种因素导致的副胰导管(圣托里尼导管)和小乳头的原发性或继发性阻塞[1] [7]。长期饮酒与该病的发生密切相关, 酒精刺激可改变副胰管内的胰腺分泌功能及副乳头功能[8], 导致副胰管引流的胰液汇入主胰管, 其形成锐角可干扰胰头顶部胰腺分泌物的流动, 导致胰液蓄积[9]增加管腔内压力, 促进胰头部假性囊肿形成, 胰液漏入沟槽区域[7]。长期饮酒也可导致胰管内胰液浓缩[10]和蛋白质沉积, 导致胰液粘稠度增加形成蛋白栓或钙化结石阻塞胰管管腔[7] [8] [11]。

此外, 过量饮酒还可促使促胰液素、缩胆囊素、胃泌素水平升高和胆碱能张力增加导致 Brunner 腺体增生, 胰液经十二指肠乳头排出受阻引起胰背部胰液沉积[3] [4] [10]。其他如十二指肠壁异位胰腺[12]、胃切除术、消化性溃疡和胆道疾病史[3]也是引起 GP 的重要因素。GP 发展的最终共同途径似乎与胰管阻塞、活化蛋白水解酶外渗引发胰腺炎以及级联的慢性炎症和纤维化相关[13]。各种因素致使胰管阻塞形成胰管内高压导致腺泡细胞内钙离子水平显著上升, 溶酶体提前激活胞内酶原活化消化胰腺自身从而释放多种炎症介质如肿瘤坏死因子- α (TNF- α)、白介素-1 (IL-1)、花生四烯酸代谢产物、活性氧(ROS)等增加血管通透性导致大量炎性渗出; 此外补体系统的激活也会放大炎症反应增加组织损伤。众多因素以正反馈形式相互作用使炎症逐级放大, 导致胰腺组织产生持续的炎症反应及纤维化。

GP 多见于有饮酒及吸烟史的中年男性[14] [15], 其临床表现与慢性胰腺炎类似[16], 常见有上腹部疼痛、恶心和反复出现的餐后呕吐。上述症状会持续几周到几年, 从而导致体重减轻。累及胆总管和炎症后狭窄的患者可表现波动性黄疸[17]。此外, 胰腺内外分泌功能受损时会出现血糖异常、脂肪泻等相关症状[15]。

GP 在诊断方面亦尚未建立统一的标准。实验室检查可能显示胰腺酶(淀粉酶和脂肪酶)轻微或显著升高, 部分患者可见白细胞升高, 偶可见肝酶升高, 而肿瘤标记物 CEA 和 CA19-9 通常是正常的[13]。孤立性淀粉酶或脂肪酶升高也有报道, 其淀粉酶水平可高达 40,000 U/L [18]。此外, 文献中也有关于高水平的 CA19-9 [19]和癌胚抗原[18]的报道。虽然仅靠影像学不能明确诊断 GP, 但一些放射学特征有利于该病的鉴别。超声显示十二指肠内壁增厚, 囊性间隙及沟内片状低回声区[7] [20]。腹部 CT 表现为胰头和十二指肠壁之间出现低密度、低强化的团块影, 动态增强检查晚期延迟强化, 十二指肠壁增厚、肠腔狭窄, 壁内和沟槽区可见小囊肿[10] [21]。MRI 显示沟槽区片状团块影, 相对于胰腺实质, T1 加权像表现为低密度信号影[1] [7], T2 加权像在病变早期由于局部水肿、瘢痕改变存在表现为高密度信号影, 病变晚期因纤维化改变表现为低密度信号影[7] [20] [22]。因此, 根据 T2 加权像在不同发病时间表现出不同程度的强化可一定程度上提示病情活动度[21]。磁共振胰胆管成像(MRCP)作为一种无创检查可清晰显示胰胆管形态及走行。单纯型 GP 胰管、胆总管形态及走行基本正常, 而节段型 GP 可见胆总管及主胰管远端狭窄, 近端扩张。此外, 十二指肠壁增厚及占位性病变可导致十二指肠与远端导管之间的距离增加[1] [7]。相比恶性肿瘤导致的胆总管不规则局灶性狭窄, GP 导致的狭窄通常是规则光滑且逐渐变细[1] [19] [21]。纤维胃镜可见十二指肠局部粘膜充血水肿, 降段狭窄伴有息肉样改变[4] [23]。超声内镜(EUS)则显示十二指肠球部及降部壁内增厚及囊肿形成[23], 胆总管轻度狭窄[20]。

由于临床表现、实验室及影像结果经常重叠, GP 在诊断上容易造成误诊。其鉴别诊断包括胰腺癌、其他胰腺肿瘤、壶腹周围癌、胃肠道间质瘤及累及沟区的急性水肿型胰腺炎等[25]。其中由于胰头肿块样增大可掩盖胰沟受累情况, 因此 GP 很容易与胰头癌混淆[25]。此外, 有文章报道存在同患 GP 及胰腺癌的情况[23] [26]。胰腺癌与节段型 GP 的鉴别点如下: GP 患者多有长期饮酒史, 而胰腺癌患者与饮酒无明显相关; 与胰腺癌患者升高情况相比, GP 患者的肿瘤标志物(CEA, CA19-9)通常在正常范围; GP 可见胰腺周围血管被推压移位, 但血管通常完整, 而胰腺癌常见血管包绕浸润[1] [7] [27]; 此外, 沟槽区延迟强化, 十二指肠壁增厚及囊性变伴肠腔狭窄, 胆总管远端渐进性狭窄等 GP 典型影像学表现也是与鉴别胰腺癌的重要特征。

目前 GP 的治疗方法主要包括内科保守治疗、内镜治疗及手术治疗, 其症状完全缓解有效率分别为 50%、57%、79% [16]。近年来有学者提出 GP 的“阶梯式”疗法, 即根据病情严重程度按照“内科保守治疗-内镜治疗-手术治疗”依次进行[28] [29]。内科保守治疗以戒烟戒酒等改善生活方式为基础, 辅以抑制胰酶分泌、镇痛药物治疗[7], 必要时采用肠外营养支持[7] [28]。内镜治疗包括胆汁或胰液引流、支架植入/扩张、囊肿开窗引流等[7] [28]。相比于外科手术, 内镜治疗术后并发症概率较低且严重程度不高

[28]。当经内科保守治疗症状无明显改善或高度怀疑恶变时首选 Whipple 手术治疗[7] [30]-[32]。手术治疗能明显改善患者疼痛及体质量减轻等主要症状, 并可对手术标本进行病理检查排除恶性肿瘤可能[14] [33]。有文献报道 Whipple 术后有 76% 的患者疼痛可获得完全缓解, 1 年后体重基本恢复正常水平[34]。Ooka 等[35]一项回顾性分析显示纳入研究的所有 GP 患者无一例出现器官衰竭, 且在平均 5 年的随访期中仅 14.6% 的患者需行手术治疗, 提示该病内科保守治疗预后相对良好。

本病例中, 患者为中青年男性, 主要临床症状为腹痛、恶心、食欲不振及体质量下降, 有长期吸烟史, 影像学提示十二指肠降段及胰头病变, 不易与胰腺癌、异位胰腺、壶腹周围癌及其他胰腺肿瘤等疾病相鉴别。入院后查肝功能指标、血 IgG、肿瘤指标均正常, 暂不支持胰腺恶性肿瘤及自身免疫性胰腺炎诊断。依据生化检查、胃镜、超声内镜及病理活检提示炎症性病变可能性大, 加之相对典型的影像学特点综合考虑诊断为沟槽状胰腺炎。

4. 结论

综上, GP 是一种罕见的局限性慢性胰腺炎, 预后相对良好, 临床症状无明显特异性, 易引起误诊, 其相对典型的影像学表现是正确诊断的关键。对于初治或轻症患者在充分排除恶性病变的基础上应首选保守治疗以避免不必要的早期手术干预。

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