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罕见病例

小肠巨大炎性纤维性息肉合并肠套叠 1 例并文献复习

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【摘 要】 报道 1 例小肠巨大炎性纤维性息肉合并肠套叠患者的临床资料, 并进行文献复习。

【关键词】 炎性纤维性息肉; 肠套叠; 小肠肿瘤; 多学科诊疗

【中图分类号】 R574.5

【文献标识码】 B

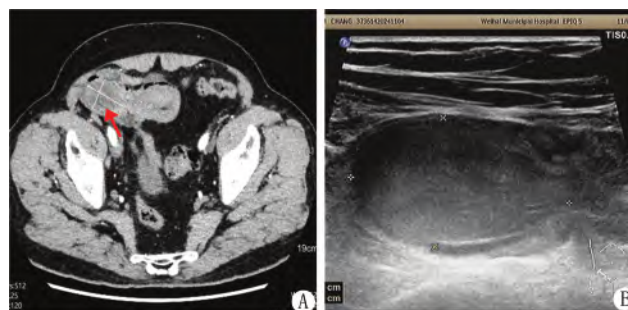
A case of giant inflammatory fibrous polyp of the small intestine with intussusception and a literature review Peng Xiang*, Li Xiaopeng, Du Chao, Chu Yanliu. * School of Clinical Medicine, Shandong Second Medical University, Shandong, Weifang 261000, China/Department of Gastrointestinal Surgery, Weihai Municipal Hospital, Shandong, Weihai 264200, China
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【Abstract】 A case of giant inflammatory fibroid polyp of the small intestine complicated with intussusception is reported and a literature review is provided.

【Key words】 Inflammatory fibroid polyp; Intussusception; Small intestinal tumors; Multidisciplinary management

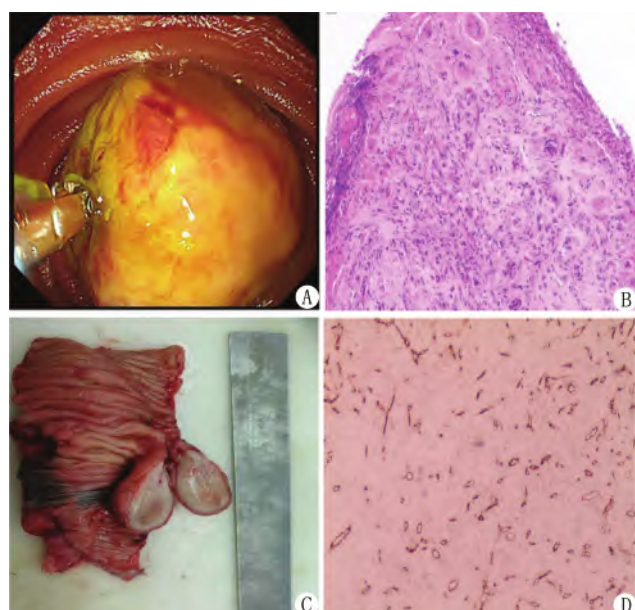
患者,男,74岁,因“腹痛 15 d”于 2024 年 11 月 2 日入院。15 d 前患者无明显诱因出现腹痛,以脐周为主,为阵发性隐痛,自觉饭后加重,无其他明显不适。既往高血压、冠心病 10 年余,9 年前行冠状动脉支架植入术,否认药物过敏史,否认家族遗传病史。无吸烟史,饮酒史 50 余年,每天约 150 g。查体: T 36.2℃, P 76 次/min, R 16 次/min, BP 168/86 mmHg。神志清楚,精神不振,结膜轻度苍白,心肺听诊未见明显异常,腹软,脐周压痛,无反跳痛,肠鸣音正常,双下肢无水肿。血常规示 RBC $4.10 \times 10^{12}/L$, Hb 80.00 g/L; 便常规示大便隐血试验阳性。腹部 CT(图 1A)示右下腹局部小肠肠壁增厚,增强扫描可见强化。腹部彩色超声(图 1B)提示第 5~6 组小肠内可见多发带蒂肿物。影像学诊断为肠套叠并肠壁水肿,考虑第 5~6 组小肠多发腺瘤样息肉,性质待定。为明确肠套叠情况和息肉性质,于 11 月 7 日行气管插管全麻小肠镜检查(图 2A):经肛侧进镜,通过回盲瓣继续进镜约 200 cm 可见巨大肿物,大小为 4.5 cm×4.5 cm,堵塞管腔,无法继续进镜,活检肿物组织 6 块,触之质硬,易出血,并行纳米碳标记。活检组织行病理检查(图 2B):灰白色组织 6 块,直径 0.1~0.2 cm,间质血管增生伴玻璃样变性,其间散在梭形细胞及炎性细胞浸润,另见炎性坏死渗出组织。由于患者肿物巨大,小肠镜下无法切除,经多学科讨论,患者有手术指征,遂于 11 月 11 日在全麻下行腹腔镜小肠肿物切除术,术中探查示肿瘤距离屈氏韧带 200 cm,大小 3.0 cm×3.0 cm,质硬,肿物套叠入远端小肠形成梗阻,近端肠管扩张、水

肿。将距离肿瘤 10 cm 远端及近端小肠完整切除。术后病理结果:肿瘤大小为 4.5 cm×2.8 cm×2.8 cm,基底宽 1.9 cm,位于黏膜及黏膜下层,肿物切面灰白、灰红色,质稍韧(图 2C)。免疫组化染色: CD117(-), Dog-1(-), CD34(血管+), SMA(血管壁+), S-100(-), Ki-67(5%+), Desmin(-), CD21(-)(图 2D)。结合病理学检查结果,最终诊断为炎性纤维性息肉(inflammatory fibroid polyp, IFP)。术后患者腹痛明显减轻,复查腹腔超声未见明显肿块及积液。患者 1 年内门诊定期复查均未见复发。



注: A.腹部 CT 示右下腹局部小肠壁增厚,增强扫描可见强化,小肠内可见一团块占位,大小为 4.3 cm×3.1 cm,管腔狭窄阻塞; B.腹部彩色超声示第 5~6 组小肠内可见多发带蒂肿物,较大为 4.4 cm×2.8 cm,边界清,形态规整,呈均匀低回声,与黏膜层相延续。

图 1 患者腹部 CT 及超声影像学表现



注: A.小肠镜检查示肿物质硬,易出血; B.活检回肠肿物病理示间质血管增生伴玻璃样变性,其间散在梭形细胞增生及炎性细胞浸润,另见炎性坏死渗出组织(HE 染色 $\times 40$); C.小肠肿物切面灰白、灰红色,质稍韧,内无明显出血坏死,位于黏膜及黏膜下层,手术切缘未见肿瘤组织; D.免疫组化结果($\times 40$): CD34(血管+).

图2 患者小肠镜及活检组织病理表现

讨论 炎性纤维性息肉(IFP)属于罕见的良性间叶组织来源肿瘤,其发病高峰多集中于50~60岁,无性别差异^[1]。通常情况下,IFP多为单发,约76%的患者病灶位于胃窦部,仅有约20%的患者病灶来源于小肠,而来源于食管、结直肠的情况极为少见。该肿瘤常起源于黏膜层及黏膜下层,极少对肌层造成侵犯^[2]。相关文献指出,IFP最初被认为是由机体应激反应所产生的非肿瘤性病变^[3],后有观点认为其起源于树突状细胞^[4]。现发现血小板衍生生长因子受体A(PDGFR α)的基因激活突变可能是重要的发病机制之一^[5]。小肠部位IFP患者可能呈现腹痛、腹泻、肠梗阻及消化道出血等临床表现。当息肉体积较大或者出现肠道狭窄时,可引发肠梗阻或肠套叠等严重并发症。多数患者无症状,仅在体检时发现。目前,内镜下切除[如内镜下黏膜切除术(EMR)、内镜黏膜下剥离术(ESD)]是较小病灶(直径<2 cm)的常规治疗方式;而对于较大病灶所引发的严重并发症如肠梗阻且术前病理无法明确诊断的IFP患者,则需要外科手术切除病变肠管^[6]。

本例患者因腹痛15 d 来诊,入院后行血常规检测提示贫血,结合影像学检查诊断肠套叠及小肠息肉可能,且不能排除小肠癌,故采取小肠镜检查并取活检。病理检查显示病变位于回肠,累及黏膜及黏膜下层,且肿物巨大,经多学科讨论,排除手术禁忌证后,通过腹腔镜切除肿物,术后病理最终诊断为小肠IFP。IFP为良性肿瘤,预后良好,术后复发率低。少数病例可能复发,但通常不转移^[7]。

IFP的典型白光内镜表现为直径多<2 cm的孤立隆起,表面可伴糜烂或溃疡,根据隆起形态及其与周边黏膜的边界清晰

度,可参照山田分型分为4型^[8]。超声内镜(ultrasound endoscopy,UES)下,IFP多表现为起源于黏膜下层的低回声病灶,边界清晰,少数可累及黏膜肌层^[7]。UES可明确病灶起源与浸润深度,结合光学内镜表现,指导后续治疗。例如,起源于黏膜下层、带蒂的息肉样病灶(山田Ⅲ/Ⅳ型)可采用EMR,对于较大(>2 cm)、广基的病灶(山田Ⅰ/Ⅱ型),则可选择ESD^[9]。然而,内镜下IFP表现并无特异性,极易误诊为胃肠道间质瘤(gastrointestinal stromal tumor,GIST)或癌(如本例术前疑诊小肠癌)^[10],因此仅依靠影像学及内镜下检测难以明确其诊断,需综合其组织学特征、免疫表型及基因检测结果进行判断。具体而言:首先,在病理组织学上,胃IFP常具有代表性的“洋葱皮样结构”组织学特征,而肠IFP常缺乏此表现^[11];其次,免疫表型以CD34(+)、CD117(-)、DOG-1(-)为核心鉴别标志^[12];最后,约55%的病例存在PDGFRA突变(胃多见于18号外显子,肠多见于12号外显子),而无KIT基因突变^[13]。

综上所述,IFP在临床上少见,且影像学及内镜下表现缺乏特异性,在诊治过程中存在挑战。尤其当病变位于小肠时,其解剖位置深在、内镜操作及深部活检受限,进一步增加了诊断难度。该病例的诊治经验表明,对于小肠部位发生的IFP,可通过多学科协作诊治(MDT)实现影像定位、内镜标记、病理鉴别与手术规划,以显著提升小肠IFP的精准诊治水平,从而避免过度治疗及医疗资源的浪费。

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