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文献综述 REVIEW

IgA血管炎发病机制及胃肠道表现的研究进展

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背景资料
IgA血管炎为累及小血管的白细胞脆裂性血管炎, 一般在儿童患者中多见, 然成人患者亦不少, 多发于秋冬季节, 可累及全身多系统, 主要临床表现分为单纯型、腹型、关节型、肾型、混合型及其他.

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Pathogenesis and gastrointestinal manifestations of IgA vasculitis

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Abstract

Immunoglobulin A (IgA) vasculitis is a form of leukocytoclastic vasculitis of small vessels, clinically characterized by purpuric skin lesions unrelated to any underlying coagulopathy, gastrointestinal manifestations, arthritis and/or arthralgia and renal involvement. The etiology remains unknown, but various triggers including infections, vaccination, drugs and malignancy have been hypothesized to be associated with the development of IgA vasculitis. Although the pathogenesis has not been completely figured out, genetic predisposition, aberrant glycosylation of the hinge region of IgA1, activated complements, cytokines and chemokines were put out to play important roles in the immunopathogenesis of IgA vasculitis. Histologically, the infiltration of small blood vessels with polymorphonuclear leukocytes and the presence of leukocytoclasia are typical pathologic findings in IgA vasculitis. The treatment is usually supportive, and advanced treatments include immunosuppressive drugs (glucocorticoids and immunosuppressive agents), hemopurification and surgery. The prognosis depends on the age at the disease onset and the renal involvement or not.

Key Words: IgA vasculitis; Gastrointestinal manifestation; Pathogenesis; Endoscopy; Treatment

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摘要

IgA血管炎是累及小血管的白细胞脆裂性小血管炎, 主要临床表现为与凝血功能无关的皮肤紫癜性病变、胃肠道表现、关节炎或关节疼痛、肾脏病变. 病因目前尚不明确, 可能与感染、疫苗、药物及肿瘤性疾病等相关. 发病机制目前考虑与患者自身遗传易感性、IgA1免疫球蛋白“O”铰链区糖基化异常、补体系统异常激活、炎性因子与促炎性因子和自身抗体的产生等方面密切相关. IgA血管炎病理学上典型表现为多形核白细胞渗出及白细胞脆裂性血管炎. IgA血管炎治疗分为对症支持治疗、免疫抑制治疗(糖皮质激素和免疫抑制剂)、血液净化治疗及外科手术治疗. IgA血管炎预后与发病年龄及是否累及肾脏相关.

关键词: IgA血管炎; 腹型; 发病机制; 内镜检查; 治疗

核心提示: 本文陈述了腹型IgA血管炎的发病机制、临床特点、内镜下改变及治疗方案.

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0 引言

IgA血管炎[1,2], 曾被命名为过敏性紫癜, 为IgA1免疫复合物沉积在血管壁而引起一种系统性血管炎, 一般累及小血管. 该病首次在1801年由Heberden教授进行了详细的阐述, 随后Schonlein教授在1837年揭示了紫癜与关节炎的联系并命其名为“风湿性紫癜”, 后来在1874年, 其学生Henoch教授进一步发现并详细阐述了紫癜与胃肠道症状的关系, 并在1899年报道该病还可累及肾脏[3]. IgA血管炎主要累及皮肤、关节、胃肠道、肾脏, 引起皮肤紫癜, 关节炎或关节肿痛, 腹痛、消化道出血, 血尿、蛋白尿、肾功能不全等临床表现, 其他少见的还可累及心脏、眼睛、肺脏[4]、睾丸[5]及中枢神经系统[6]等.

1 一般情况

IgA血管炎在儿童患者中多见, 各家报道其年发病率从3/10万-26.7/10万不等[7-9], 在3-12岁儿童中尤为多见[10], 成人发病率则相对较低, 年发病率从为0.8/10万至1.8/10万不等[10-12], 平均发病年龄为45-50岁[10,12,13]. 发病年龄最小者为6 mo[14], 最大为86岁[15]. 很多研究结果均表明在IgA血管炎患者中, 男性略多于女性[8,13]. IgA血管炎一般多发于秋、冬季节, 夏季最少见[16,17].

2 病因

IgA血管炎病因尚不明确, 但其多于秋冬季节发病且大部分患者起病前有上呼吸道感染的病史[18-20]的特点均提示感染为引起IgA血管炎重要的病因, 这些致病菌包括Weiss等[21]提出的微小病毒B19、乙型肝炎病毒、丙型肝炎病毒、腺病毒、A组β溶血性链球菌、金黄色葡萄球菌、支原体, 幽门螺杆菌[22,23], 还有另外一些原因包括药物的使用[24]、肿瘤性疾病[25-27]、接种疫苗[28]等均可能导致IgA血管炎的发生.

3 发病机制

IgA血管炎的发病机制并不完全清楚, 目前认为其发病与患者自身遗传易感性、IgA1免疫球蛋白“O”铰链区糖基化异常、补体系统异常激活、炎性因子与促炎性因子和自身抗体的产生等方面密切相关[29,30].

3.1 遗传易感性 研究[31]发现, *HLA-DRB1*基因及其等位基因, *HLA-B35*基因[32]、*HLA-DQA1*基因[33]、*HLA-A*基因(*HLA-A1101*、*HLA-A2601*)和*HLA-B*基因(*HLA-B1501*、*HLA-B3503*、*HLA-B52*、*HLA-B07*、*HLA-B40*)[34]均与IgA血管炎的发生及发展密切相关, 此外, 一些炎性因子及RAS相关的靶基因正越来越成为研究的热点.

3.2 IgA1铰链区糖基化异常 IgA分为IgA1(80%-90%)和IgA2两种亚型, 其中IgA1的重链区铰链(O)上分布着5-6个糖基化位点, 此为其区别于IgA2和其他免疫球蛋白的重要标志[35-37]. IgA1铰链区糖基化异常, 使其不能被肝细胞识别清除, 更易于自我聚集或形成抗原抗体高分子免疫复合物, 并在系膜区或内皮下沉积, 从而引起小血管内皮细胞的损伤, 引起白细胞脆裂性血管炎[38,39].

相关报道

Zhang等回顾性分析总结了腹型IgA血管炎的临床特点及内镜下改变, 指出内镜检查在腹型IgA血管炎诊治过程中的重要作用.

创新盘点

本文阐述了腹型IgA血管炎的临床表现、内镜下特点及治疗方案, 并相对全面的总结了IgA血管炎的发病机制.

3.3 补体系统 补体系统通过经典激活途径[40]、替代激活途径[41-45]、凝集素激活途径[45,46]参与IgA免疫复合物清除障碍过程[47].

3.4 炎性因子与促炎性因子 Th17细胞产生的炎性因子如IL-6、IL-17等[48]在一些促炎性因子如肿瘤坏死因子-α(tumor necrosis factor α, TNF-α)、白介素(interleukin, IL)-1β、IL-2、IL-6、转化生长因子β(transforming growth factor-β, TGF-β)、血管内皮生长因子(vascular endothelial growth factor, VEGF)等[49-53]刺激血管内皮细胞产生的趋化因子的作用下, 被诱导黏附在血管壁, 作用于血管内皮细胞从而引起血管炎症性改变[54,55].

3.5 自身抗体 抗磷脂抗体[56,57]、狼疮抗凝物抗体[58,59]、抗中性粒细胞胞浆抗体(anti-neutrophil cytoplasmic antibody, ANCA)[60]、抗核抗体和IgA类风湿因子[61,62]、IgA抗内皮细胞抗体[63]等在IgA血管炎的不同发病环节中发挥着一定的作用, 但其具体作用机制尚不明确.

4 临床表现

腹型IgA血管炎临床表现多样, 包括腹痛、消化道出血(多表现为黑便、粪潜血为阳性)、恶心呕吐、腹泻、呕血等, 其发生率在成人患者与儿童患者之间并无很大差别[24,64]. 腹痛是腹型患者中最常见的临床表现[65-67], 常见腹痛部位为脐周及上腹部, 弥漫性全腹痛少见. 还有少数患者表现为转移性右下腹痛, 导致该病易误诊为急性阑尾炎[67]. 腹痛常为持续性的绞痛, 进食后加重, 这与肠道局部缺血及肠绞窄不无关系[68,69].

消化道出血如黑便、粪隐血试验阳性等往往伴随腹痛症状一起出现, 出血量一般不多, 极少会出现消化道大出血而致出血性休克需要急诊内镜或外科干预的情况[70-72].

若上述症状得不到及时缓解, 病情可进一步加重而出现肠套叠、肠梗阻、肠穿孔、肠瘘等外科急症而需内镜或手术干预治疗[68,69,73]. 一般而言, 肠套叠多见于儿童患者[74-76], 多表现为腹部可触性包块. 其按发生部位可分为回回型(51%)、回结型(39%)和空空型(7%)[69]. 回回型、空空型肠套叠因其位置较高, 灌肠效果不理想, 如24 h之内不能自行缓解, 则需外科手术处理[77]. 回结型肠套叠80%-90%为自发性肠套叠, 多因肠道远端淋巴组织异常增生引起[76], 多见于2岁以下小儿, 若2岁以上患者出现回结型肠套叠, 需警惕腹型IgA血管炎所致肠套叠[8], 该型肠套叠可通过空气灌肠或钡剂灌肠缓解[78]. 肠套叠与肠穿孔密切相关. 肠穿孔部位多为回肠, 且多发生在使用激素治疗2 wk以内[79], 可能与激素的应用掩盖了真实病情且加重了感染有关[80], 往往在肠穿孔之前会出现病情好转的假象[81]. 肠梗阻多发于十二指肠球部和/或回盲部[78,82-84], 与胃肠道局部缺血相关[85].

其他少见的并发症还包括蛋白丢失性肠病[86]、脂肪泄[87]、肠瘘[73]、壁间血肿[88]、胆囊炎[89,90]、浆膜炎[91,92]、乳糜腹水[93]、梗阻性黄疸[94]等.

5 检查

5.1 内镜检查 内镜下胃肠道黏膜病变通常表现为弥漫性黏膜充血水肿、瘀点瘀斑、糜烂出血点、溃疡性病变、结节样改变等[95-97], 少数患者表现为病变部位梗阻或过度狭窄以至内镜无法通过. 其中溃疡性病变多为形态不规则小溃疡, 基底面积0.2-1.0 cm^2不等, 基底干净. 十二指肠降部、末端回肠分别为胃镜、肠镜检查中最常见的病变部位[97,98], 且这两个部位的病变表现通常也最严重. 超声内镜下病变部位一般表现为黏膜及黏膜下层水肿[94]. 但胃镜及肠镜所能看到的范围有限, 在胃镜及肠镜检查结果均阴性时, 胶囊内镜因其拥有纵观消化道全程、检查无痛苦的优势, 对腹型IgA血管炎的诊断很有帮助[99], 且对治疗也有一定的指导意义[100]. 需要注意的是, 对于胃肠道蠕动功能减弱、可能合并肠梗阻的患者胶囊内镜忌用[99].

5.2 彩色多普勒超声 彩色多普勒超声可发现病变部位肠壁增厚(多为对称性), 肠壁回声均匀减低, 肠腔狭窄, 黏膜皱襞不清、肠道蠕动减弱甚至消失, 呈“腊肠”样表现. 长轴面显示肠管呈管状狭窄, 横断面显示圆形低回声肿块, 肠腔呈向心性或偏心性狭窄[101]. 还可发现腹腔积液、肠套叠(呈典型的“同心环”征和“肾”征)[102-104]. 彩色多普勒超声检查通过红、蓝血流信号可判断病变肠管血供情况, 对疾病严重程度的判断有一定的帮助[105].

5.3 CT扫描 CT扫描能发现多部位的肠道病变, 主要位于空肠和回肠[106], 与其血管分布相对较

多的解剖学特点相关. 表现为肠壁节段性增厚, 肠腔呈不对称性狭窄. 由于损伤血管的渗出液主要集中在肠道黏膜下层, 增厚的肠壁表现为密度减低, 增强后较正常肠壁强化程度低, 故呈典型“靶征”表现. 受累肠管周围的血管充血肿胀, 淋巴结增多增大, 部分可伴有腹腔积液[106,107]. 此外, CT检查还能显示腹型IgA血管炎较常见的并发症如肠套叠、肠穿孔及肠梗阻. 肠套叠CT影像学特点: 当扫描层面与套叠肠管平行时, 肿块内呈分层状, 高低密度相间, 其间可见肠系膜脂肪影; 当扫描层面与套叠肠管垂直时, 肿块内呈同心环状[101]. 肠穿孔即显示为膈下可见游离气体密度影. CT扫描不仅可显示梗阻扩张的肠管, 同时还可以显示造成梗阻的狭窄段肠管.

5.4 腹部平片 腹部平片对于怀疑腹型IgA血管炎合并肠梗阻、肠穿孔等并发症的患者是首选检查, 能通过是否有肠管扩张及液气平面、是否可见膈下气体密度影快速确认是否合并上述急腹症.

5.5 实验室检查 Kimura等[108]在研究中曾指出腹型IgA血管炎疾病过程中IL-6及IgM型抗磷脂酰丝氨酸-凝血酶原复合物抗体(antiphosphatidylserine-prothrombin complex antibody, anti-PSPT)水平升高, 而ASO、CRP、IL-8、抗心磷脂抗体(anticardiolipin antibody, aCL)及TNF-α水平与腹型IgA血管炎缺乏明确的关系. 另有报道[109]提出部分腹型IgA血管炎患者可表现为血清IgA水平升高. Prenzel等[110]曾指出Ⅷ因子活性减低与腹型IgA血管炎严重程度相关. 还有一些指标与腹型IgA血管炎相关如血小板计数增高、白细胞计数增多、血小板平均体积减小、嗜酸性粒细胞减低、血沉增快等, 但均缺乏特异性[110-112].

6 病理

典型胃肠道病变部位活检标本表现为血管壁红染、纤维素样坏死、脆裂, 管腔内血栓形成, 红细胞溢出, 符合白细胞脆裂性血管炎[113,114], 免疫组织化学染色可见IgA颗粒样沉积于血管壁.

7 诊断及鉴别诊断

7.1 诊断标准

7.1.1 1990年诊断标准: 1990年美国风湿病学会(ACR)诊断标准[115]为具备以下2条或2条以上: (1)与凝血功能无关的可触性皮疹; (2)发病年龄≤20岁; (3)急性腹痛; (4)组织活检示小动脉小静脉粒细胞浸润.

7.1.2 2006年诊断标准: 2006年欧洲抗风湿病联盟和欧洲儿科风湿病学会(EULAR/PReS)[116]制定了新的诊断标准: 可触性(必要条件)皮疹伴以下任何一条: (1)弥漫性腹痛; (2)任何部位活检示IgA沉积; (3)关节炎和/或关节痛; (4)肾脏受损表现(血尿和/或蛋白尿).

7.1.3 2012年更名: 2012 Chapel Hill年会议(International Chapel Hill Consensus Conference, CHCC)在新的血管炎分类标准中将过敏性紫癜(henoch-schonlein purpura, HSP)改名为IgA血管炎[2].

7.2 鉴别诊断

7.2.1 IgG4相关性疾病: IgG4相关性疾病[117]累及消化系统时可出现腹痛、恶心呕吐等临床症状, 有时与腹型IgA血管炎引起的胃肠道表现不易鉴别, 但前者有其自身疾病特点: 一个或多个器官或组织似肿瘤性的肿胀增大; IgG4阳性淋巴细胞大量增生, 在组织中浸润; 血清IgG4细胞水平显著增高; 对糖皮质激素反应良好.

7.2.2 系统性红斑狼疮: 系统性红斑狼疮[118]患者约30%出现食欲减退、腹痛、呕吐、腹泻等消化系统症状, 少数可并发急腹症如胰腺炎、肠坏死、肠梗阻等, 需注意与腹型IgA血管炎鉴别. 前者血清中可以查到多种自身抗体如抗核抗体、抗双链DNA抗体、抗ENA抗体、抗磷脂抗体等, 并伴其他系统表现如皮肤表现(颊部红斑、盘状红斑)、关节表现(关节炎)、肾脏病变、神经系统等.

7.2.3 急性阑尾炎: 急性阑尾炎患者多有转移性右下腹痛史, 胃肠道症状突出, 右下腹麦氏点有固定压痛, 腹膜刺激征阳性. 随病情进展, 腹部压痛及肌紧张范围可加大.

7.2.4 急性肠系膜淋巴结炎: 急性肠系膜淋巴结炎常常先表现为以发热为主的上呼吸道感染症状, 继而出现腹痛, 主要表现为脐周或右下腹钝痛, 体检时腹痛部位随体位而变化, 不伴腹肌紧张, 抗感染治疗短期内腹痛可减轻、缓解.

腹型IgA血管炎患者腹痛多表现为脐周或下腹绞痛或钝痛, 进食后加重, 无腹肌紧张, 对糖皮质激素反应佳.

■应用要点
糖皮质激素及免疫抑制剂治疗可以明显改善腹型IgA血管炎的临床症状, 必要时可进一步行血液净化治疗.

名词解释

腹型IgA血管炎: 指临床上以胃肠道表现为主的IgA血管炎, 主要表现为腹痛、消化道出血等, 内镜下表现为弥漫性黏膜充血水肿、瘀点瘀斑、糜烂出血点、溃疡性病变、结节样改变等. 十二指肠降部及回盲部为常见累及部位, 激素治疗有效.

8 治疗

IgA血管炎具有一定的自愈倾向, 且症状、病变程度及受累系统差异较大, 其具体治疗方案应遵循个体化原则. IgA血管炎的治疗总体上分为对症支持治疗、免疫抑制治疗、血液净化治疗及外科手术治疗.

8.1 对症支持治疗 对于病变程度轻微、受累系统少的患者可仅予以对症支持处理, 包括缓解关节疼痛、腹痛及胃肠道出血等临床表现. 对于常规处理胃肠道症状缓解不满意的患者, 可进一步考虑免疫抑制治疗及血液净化治疗.

8.2 免疫抑制治疗 免疫抑制治疗包括糖皮质激素治疗及免疫抑制剂治疗. Ronkainen等[119]、Jauhola等[120]分别通过研究证实应用糖皮质激素2 wk之内可减轻腹痛程度, 并能缩短腹痛病程. 《儿童过敏性紫癜询证诊治建议》[109]中指出对有腹痛症状患者推荐使用激素治疗: (1)口服泼尼松治疗, 1-2 mg/kg(最大剂量60 mg) 1-2 wk, 后1-2 wk减量. 胃肠道症状较重(持续腹痛、胃肠道出血、肠系膜血管炎及胰腺炎等)不能口服药物治疗者推荐使用静脉激素治疗, 一般剂量为氢化可的松琥珀酸钠5-10 mg/(kg•d), 根据病情可间断4-8 h重复使用; (2)甲泼尼龙5-1 mg/(kg•d)[病情严重者如肠系膜血管炎大量出血者给予激素冲击治疗量可达15-30 mg/(kg•d), 最大剂量<1 g/d, 连用3 d, 必要时1-2 wk后重复冲击3 d]; (3)或地塞米松0.3 mg/(kg•d), 严重症状控制后应改口服糖皮质激素, 并逐渐减量, 总疗程推荐2-4 wk, 注意疗程不宜过长. 然而, 糖皮质激素的应用并不能阻止IgA血管炎进一步累及肾脏, 也不能降低对腹型IgA血管炎发生肠套叠、肠梗阻、肠穿孔等胃肠道并发症的发生率[120-123]. 常用的免疫抑制剂包括环磷酰胺、秋水仙碱、氨苯砜、硫唑嘌呤、吗替麦考酚酯、环孢素A、利妥昔单抗等, 其中常用于治疗腹型IgA血管炎的免疫抑制剂为环磷酰胺[124]和氨苯砜[125].

8.3 血液净化治疗 对于激素和免疫抑制剂治疗效果不理想的腹型患者, 可考虑行血液净化治疗: 血浆置换[126]、血液灌流[127]等, 通过清除体内异常免疫球蛋白、抗原抗体免疫复合物、自身抗体、炎性因子等而减轻异常免疫反应, 从而缓解临床症状.

8.4 外科手术 对于肠梗阻、肠穿孔等内科保守治疗无效的患者, 需及时行外科手术治疗, 以免耽误最佳治疗时机.

9 预后

IgA血管炎多为自限性疾病, 一般预后良好. 腹型IgA血管炎临床表现中症状较重的情况如消化道出血、肠套叠、肠穿孔、肠梗阻、肠瘘等影响患者近期预后, 需及时的识别及处理, 否则会耽误患者病情, 甚至危及患者生命. 而起病年龄<20岁、肾脏受累、紫癜持续超过1 mo则为影响IgA血管炎远期预后的危险因素[64,128,129].

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同行评价

本文综述分析了腹型IgA血管炎的发病机制、临床表现和临床治疗, 有较好的学术水平.

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