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·专家述评·

重视对髓鞘少突胶质细胞糖蛋白抗体阳性视神经炎的再认识

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[摘　要]　视神经炎(optic neuritis,ON)是指视神经的炎性脱髓鞘病变，是引起中青年人视力下降的主要原因。近年来，髓鞘少突胶质细胞糖蛋白抗体阳性视神经炎(myelin oligodendrocyte glycoprotein antibody-positive ON,MOG-ON)成为神经眼科领域的研究热点，国内外报道不断增加。2021年3月，中华医学会眼科学分会神经眼科学组制定了《中国脱髓鞘性视神经炎诊断和治疗循证指南(2021年)》，将MOG-ON作为新的视神经炎亚型纳入脱髓鞘性视神经炎的诊疗体系，给广大眼科医生提供了新的参考依据。因此，临床医生需要充分认识MOG抗体相关疾病和MOG-ON的临床特征和治疗进展，努力提高其诊断和治疗水平，使此类患者能够得到更多的获益，造福于更多的视神经疾病患者。

[关键词]　髓鞘少突胶质细胞糖蛋白抗体；视神经炎；临床特征；治疗进展

电子全文

Pay attention to the re-understanding of myelin oligodendrocyte glycoprotein antibody-positive optic neuritis

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Abstract Optic neuritis(ON)is an inflammatory demyelinating disease of the optic nerve, which is the main cause of vision loss in young and middle-aged people. In recent years, myelin oligodendrocyte glycoprotein antibody-positive ON(MOG-ON)has become a research hotspot in the field of neuro-ophthalmology, and reports at home and abroad are increasing. In March 2021, the Neuro-ophthalmology Group of Ophthalmology Branch of Chinese Medical Association formulated the Evidence-Based Guidelines for the Diagnosis and Treatment of Demyelinating Optic Neuritis in China(2021) , and included MOG-ON as a new optic neuritis subtype in the diagnosis and treatment system of myelinating optic neuritis providing a new reference for the majority of ophthalmologists. Therefore, clinicians need to fully understand the clinical features and treatment progress of MOG antibody-related diseases and MOG-ON, and strive to improve the level of diagnosis and treatment, so that such patients can get more benefits and benefit more patients with optic nerve diseases.

Keywords myelin oligodendrocyte glycoprotein antibody; optic neuritis; clinical characteristics; treatment advancement

视神经炎(optic neuritis,ON)是指视神经的炎性脱髓鞘病变，可导致急性或亚急性视力下降，是引起中青年人视功能损害的主要原因。水通道蛋白4(aquaporin 4,AQP4)抗体和髓鞘少突胶质细胞糖蛋白(myelin oligodendrocyte glycoprotein,MOG)抗体成为ON诊断必不可少的生物标志物，改变了眼科医生对于典型性ON的理解和认识[1]。近几年，MOG抗体阳性视神经炎(MOG antibody-positive ON,MOG-ON)成为神经眼科领域的研究热点，国内外报道不断增加[2-5]。2021年3月，中华医学会眼科学分会神经眼科学组制定了《中国脱髓鞘性视神经炎诊断和治疗循证指南(2021年)》[6]，将MOG-ON作为新的ON亚型纳入脱髓鞘性视神经炎的诊疗体系，给广大眼科医生提供了新的参考依据。因此，临床医生需要充分认识MOG-ON的临床特征，努力提高对该病的诊断和治疗水平，使患者能够得到更多的获益，造福于更多的视神经疾病患者。

1 MOG抗体与MOG抗体相关疾病

MOG抗原是一种1型跨膜蛋白，表达于少突胶质细胞的细胞膜上，属于免疫球蛋白超家族[7]。虽然，MOG仅占中枢神经系统(central nervous system,CNS)髓鞘蛋白成分的0.05%，但又是CNS髓鞘成分中抗原性最强的蛋白[8]。21世纪初，MOG 抗体曾被误认为是多发性硬化(multiple sclerosis,MS)的特异性生物标志物[9]，早期MOG抗体的检测方法是酶联免疫吸附实验(enzyme linked immunosorbent assay,ELISA)法，随着检测技术的发展出现了灵敏度和特异度更高的基于细胞的检测(cell-based assays,CBA)法[10]。ELISA法能识别的线性表位的MOG抗体不具有致病性，而CBA法能识别构象表位的MOG抗体具有致病性，通过CBA法检测MOG抗体证实了MS患者的MOG抗体阳性率极低，由此确定MOG抗体阳性与MS发生无直接的关系。

目前，主流观点认为MOG抗体是MOG抗体相关疾病(MOG antibody-associated disease,MOGAD)主要的生物标志物，MOGAD是一种不同于MS和视神经脊髓炎谱系疾病(neuromyelitis optica spectrum disorder,NMOSD)的独立的疾病谱[11-12]。2015年，国际NMOSD诊断标准是在MOGAD被确认为一个独立疾病实体之前制定的，约21%~42%的AQP4抗体阴性NMOSD患者的MOG 抗体呈阳性[13-14]。MOGAD的临床表型比NMOSD更加广泛，仅有约1/3的MOGAD患者符合AQP4抗体阴性的NMOSD诊断标准[15-16]。AQP4抗体和MOG抗体同时阳性NMOSD病例极为罕见，表明了NMOSD和MOGAD并非由共同的病理生物学机制介导[17]。另外，MOGAD病理标本显示的脱髓鞘病灶不伴有星形胶质细胞变性，这与NMOSD并不相同[18]。MOGAD病灶可广泛累及CNS，临床表现多样化，包括ON、横贯性脊髓炎、急性播散性脑脊髓炎(acute disseminated encephalomyelitis,AEDM)和脑干脑炎等[15, 19]，可为单一症状或者以上症状的多种组合。

2 MOG-ON临床特征

ON是MOGAD最常见的临床分型，ADEM在儿童患者中更常见。MOG-ON患者男女比例相当，临床表现多种多样，主要特征包括视盘水肿多见、容易双眼发病和复发率较高[2-3]。MOG-ON更常见于儿童患者[4-5]，与中青年患者相比，儿童患者具有不同

的临床特征，具有更好的视力恢复、更低的年复发率和更多的颅内段视神经受累[20]。MOG-ON和AQP4-ON急性期引起的视力下降通常都很严重，但MOG-ON患者的视力恢复结局明显优于AQP4-ON患者[2, 21]；仅有6%~10%MOG-ON患者的最终视力低于0.1，而AQP4-ON患者的最终视力低于0.1的比例通常超过1/3[2, 21-22]。笔者团队的一项最新研究结果显示：首次发病时有73.2%MOG-ON患者伴视盘水肿(AQP4-ON占比为38.2%)，45.0%MOG-ON患者首次发病即表现为双眼同时受累或短期内双眼先后受累(AQP4-ON占比为12.6%)；随访结束时MOG-ON患者出现严重视力下降(BCVA≤0.1)比例(15/254；5.9%)明显低于AQP4-ON(236/550；42.9%)和双抗体阴性ON组(68/218；31.2%)[5]。

部分MOG-ON患者对于糖皮质激素(激素)治疗反应敏感，但又存在着明显的激素依赖性，表现为激素减量或者停用后容易复发，这一特征符合慢性复发性炎性视神经病变(chronic relapsing inflammatory optic neuropathy,CRION)诊断标准[23-24]，而AQP4-ON患者很少出现这类情况[25]。MOG抗体阳性可以表现为短暂性或者一过性，约63%MOG-ON患者的抗体水平在发病14个月后降至检测线以下[26]，而AQP4抗体一旦阳性，通常会持续多年。MOG抗体持续阳性的患者更易复发，并且MOG抗体滴度的降低与疾病的单时相病程有关[27]。使用免疫抑制剂治疗期间，血清MOG抗体滴度可以降低或者转阴[28]。MOG抗体可以在其他CNS炎症性疾病中检测到，例如与抗N-甲基-D-天冬氨酸受体(anti-N-methyl-D-aspartate receptor,NMDAR)抗体共存[29]。

不同亚型ON的眼眶MRI检查表现存在差异性。在MOG-ON患者中，视神经增强信号可以发生在视神经周围，甚至延续到眶内组织，这在其他亚型的ON中很少见到[2, 30-31]。AQP4-ON常有长节段的视神经受累，其中视交叉和视束受累相对常见[31]。近期一项研究表明MOG-ON患者视交叉受累的比例高达16%，在这些患者中约54%存在长节段广泛的视神经增强信号[32]。由于MRI视神经受累特点不同，可以为不同亚型ON诊断及鉴别提供线索，建议对所有非典型ON患者进行眼眶MRI检查[6]。

3 MOG-ON治疗进展

目前，MOG-ON治疗研究数据有限，治疗推荐多来自一些小样本、回顾性研究，缺乏统一治疗方案，参考和借鉴专家共识和相关疾病的治疗指南具有重要意义[6, 11-33]。MOG-ON的治疗通常分为两个阶段：急性期治疗和缓解期治疗。急性期治疗的目的是最大限度地挽救视功能，防止或减少对CNS的进一步损害；维持期治疗主要目的是为了减少疾病的复发，降低失明和致残的严重程度。

大剂量甲泼尼龙冲击治疗(intravenous methylprednisolone,IVMP)是MOG-ON患者急性期的一线治疗。最近的一项回顾性研究表明，早期IVMP治疗可以改善MOG-ON患者的视力预后[34]。对于部分IVMP治疗不敏感的患者，可使用血浆置换(plasma exchange,PE)治疗。国外研究表明，对于IVMP治疗无效的MOGAD患者经PE治疗后的预后有改善[28]。笔者研究表明，PE治疗可以有效改善ON患者的视力[35]。因此，当伴有严重视力下降的MOG-ON患者经IVMP治疗效果欠佳时，PE是一种可行的治疗选择。对于IVMP治疗无反应或反应差，或病情逐渐恶化的MOGAD患者，静脉注射免疫球蛋白(intravenous immunoglobulin,IVIG)治疗有助于快速控制症状并减少复发[36]。日本学者进行的一项多中心、前瞻性、双盲、随机对照试验表明，IVIG可作为IVMP治疗不敏感ON急性期患者安全有效的治疗选择[37]。

MOG-ON患者的维持期治疗的最佳方案还不确定，期待着国内外同行开展更多的“头对头”(直接比较研究)的临床试验，筛选更优的治疗方案。多项研究表明，MOG-ON加用经典的免疫抑制治疗，包括小剂量激素、硫唑嘌呤、吗替麦考酚酯(mycophenolate mofetil,MMF)、利妥昔单抗(rituximab,RTX)和定期IVIG，可以降低疾病复发的频率，但不能完全预防复发[38-43]。一项来自13个国家29个中心的回顾性研究发现，RTX降低了MOGAD的复发率，尽管B淋巴细胞显著减少(血液中$CD19^+$B淋巴细胞比例<1%)，但仍有不少患者继续复发[44]。笔者研究发现，MMF和RTX均能降低MOG-ON患者的疾病活动程度，RTX表现出比MMF更好的耐受性；因为不加用免疫抑制治疗的MOG-ON患者中仅有不到一半会复发[45]，因此，首次发病后暂时不推荐免疫抑制剂治疗仍然是一个不错的选择。据报道，托珠单抗有助于稳定复发性MOG-ON患者的病情[46-47]，还需要开展更多的高质量临床研究来验证。最后还需要指出，传统的MS疾病修饰治疗对MOG-ON患者无效，甚至可能使患者的病情加重或恶化[40-41]。

4 展望

AQP4抗体和MOG抗体的发现加深了眼科医生对ON的认识，两种特异性抗体的检测有助于不同亚型ON患者的诊断、治疗和随访。推荐非典型ON患者进行AQP4抗体和MOG抗体筛查。针对NMOSD发病机制中的关键环节的新型的免疫靶向药物研发，比如选择性补体C5抑制剂、IL-6受体抑制剂、B淋巴细胞消耗剂，这些药物的上市已成为治疗AQP4-ON的里程碑事件。新型免疫靶向药物的成功上市为我们深入研究MOGAD发病机制提振了信心，坚信在不久的将来就会出现针对MOG-ON发病机制中的特异性靶向药物；此外，干细胞治疗、抗VEGF治疗、基因治疗可能会是潜在有效的措施，有待于更加深入的基础和临床研究；同时呼吁国内从事神经眼科的医生齐心协力、通力合作，积极开展和参与国际和国内的多中心视神经疾病的临床研究项目，尽快搭建和完善我国的神经眼科疾病数据库平台，积累更多的国人自己的循证医学证据，更好地服务于MOG-ON患者。

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