· 综述 ·

颅内段椎基底动脉夹层动脉瘤诊疗研究进展

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【摘要】 颅内段椎基底动脉夹层动脉瘤(IVBDAs)是发生于椎动脉颅内段和基底动脉的以动脉瘤形成为主要形态学改变的动脉夹层，发病原因及确切机制尚不完全明确。其临床表现不具有特异性，还可能随时间进展演变转换，影像表现复杂多变，人们对IVBDAs的流行病学、自然病史、病因及发病机制、诊断方法及治疗策略尚存有争议，现参考近年发表的文献资料，综述IVBDAs的研究进展。

【关键词】 动脉瘤，夹层；　颅内动脉瘤；　椎基底动脉；　诊断；　治疗；　综述

Advances in the diagnosis and treatment of intracranial vertebrobasilar dissecting aneurysms

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【**Abstract**】 Intracranial vertebrobasilar dissecting aneurysms (IVBDAs) are arterial dissections that occur in the intracranial and basilar arteries of the vertebral artery with aneurysm formation as the main morphological change. The causes and precise mechanism of IVBDAs are not clear. The clinical manifestations of IVBDAs are non-specific, complex and varied, and may evolve and transform over time. The imaging manifestations are complex and varied as well. There are still controversies about the epidemiology, natural history, etiology and pathogenesis, diagnostic methods and treatment strategies of IVBDAs. The paper reviews the recent literature on IVBDA and summarizes research development.

【**Key words**】 Dissecting aneurysms;　Intracranial aneurysms;　Vertebrobasilar artery; Diagnosis; Treatment;　Review

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颅内动脉夹层(intracranial artery dissections, IADs)被用于描述颅内动脉内弹力层、中膜破损,动脉壁间血肿形成的病理学状态[1]。动脉内膜破损的原因及形式多种多样,夹层发生后又受到机体自身修复机制、治疗措施及时间演变的影响[1],因此动脉夹层发生后就会进入一个动态演变的过程,不同个体和同一个体不同时期其动脉夹层的形态可以各不相同,从血管影像上可以表现为动脉狭窄、闭塞或者动脉瘤[2],一般将表现为狭窄或闭塞者称为动脉夹层,表现为动脉瘤者称为夹层动脉瘤。颅内段椎基底动脉夹层动脉瘤(intracranial vertebrobasilar dissecting aneurysms, IVBDAs)是发生于椎动脉颅内段和基底动脉的以动脉瘤形成为主要形态学改变的动脉夹层。迄今,人们对IVBDAs的流行病学、自然病史、病因及发病机制、诊断方法及治疗策略尚颇有争议,本文参考近年发表的文献资料,综述IVBDAs的诊断及治疗研究进展。

一、IVBDAs的流行病学

血管造影及尸检结果表明,IVBDAs总发病率<0.05%[3]。不同背景的研究者、不同的研究招募策略和研究方法得出了不同的研究结果,病例数在40例以上的颅内动脉夹层的报道95%以上来自亚洲[1]。基于欧美人群的研究表明,颅内动脉夹层的发病率低于颅外颈部动脉夹层的发病率,然而基于亚洲人群的研究却显示出相反的结果[4]。在椎基底动脉夹层患者出血组和非出血组的平均年龄分别为52.8岁和47.5岁,男性比例更高,而在前循环动脉夹层患者中男女比例无明显差异[5]。儿童颅内夹层动脉瘤发生概率是成年人的4倍多[6],发生于儿童时期的椎基底动脉动脉瘤大多为夹层动脉瘤[7]。随着影像学技术的发展和广泛应用,经体检发现的无症状性IVBDAs患者越来越多,故IVBDAs实际的发病率应该远高于现有的统计数据。

二、IVBDAs的自然病史和临床转归

IVBDAs的自然病史和临床转归尚不明确,是未来研究中需要重点关注的问题。有日本学者对113例未发生过卒中且未进行介入干预和抗栓治疗的IVBDAs患者进行了平均2.9年的随访发现,52例无症状患者和56例表现为疼痛的患者中仅各有1例在随访期内出现病情进展而表现为占位症状,而5例具有占位症状的患者中有1例发生在蛛网膜下腔出血并继发缺血性卒中[8]。Nasr等[9]于2018年发表了1篇关于椎基底动脉延长扩张症及梭形动脉瘤自然病史的系统评价和Meta分析显示年全因死亡率高达13%,每年有12%的动脉瘤出现体积增长,每年有3%的动脉瘤破裂出血,每年发生缺血性卒中的比率高达6%。

三、IVBDAs的发病机制

确切的病因及发病机制尚不明确,或者从本质上说IVBDAs的发生是内源性因素和外源性因素共同作用的结果。目前认为IVBDAs最常见病因为动脉粥样硬化、高血压病[10]及血流动力学因素[11]等。正常椎动脉在分出小脑后下动脉以后的位置,中膜和外膜显著变薄[12],这可能是构成IVBDAs形成的解剖学基础。滋养血管在IVBDAs的形成和发展过程中起到相当大的作用[13]。此外,还有一些少见的疾病如Marfan综合征[14]都可能IVBDAs的发生相关。近年来,日本学者提出IVBDAs可能为IgG4相关性疾病[15]。有学者提出IVBDAs可能机制为:(1)首选出现内弹力层断裂,然后代偿性内膜增生;(2)当内膜增生达到一定程度时,新生滋养血管出现;(3)新生滋养血管引起出血和壁间血肿;(4)血栓反复再通并新生血管形成进一步出血导致动脉瘤体积快速增长[16]。

四、IVBDAs的病理分型

Mizutani等[17]依据梭形夹层动脉瘤死亡患者尸检病理表现,将其分为4型:(1)1型经典型:急性广泛的内弹力层崩解且不伴有内膜增厚,1型患者的临床过程凶险,表现为急性蛛网膜下腔出血及频繁再出血。(2)2型节段扩张型:内弹力层扩展和或呈碎片状,伴内膜适度增厚,该型管腔增厚的内膜表面光滑,无血栓形成,2型患者临床过程平稳。(3)3型延长扩张型:内弹力层断裂,增厚的内膜多发夹层,管腔内血栓形成,3型大多数具有临床症状而且随着时间的推移逐渐进展。(4)4型动脉主干囊状动脉瘤:主要病理特征为少量损伤的内弹力层并且无内膜增厚,有破裂的危险。

五、IVBDAs的临床表现

临床表现不具有特异性,复杂多变,还可能随时间进展演变转换[5,18]。可以无症状或表现为非特异性的头颈部疼痛、头晕等,也可表现为急性缺血性卒中或蛛网膜下腔出血,还可表现为慢性占位及压迫症状,导致颅神经麻痹、脑干压迫症状甚至梗阻性脑积水。夹层动脉瘤导致蛛网膜下腔出血的病例占非外伤性蛛网膜下腔出血病例的3%~7%[19]。即使是表现为非出血症状的未破裂夹层动脉瘤也有

出血可能，瘤样扩张体积大、急性期双腔症及随访中增大的未破裂夹层动脉瘤易于破裂[20]。无论是否采取治疗措施，夹层动脉瘤存在较高的再出血率[5,21-22]。

六、IVBDAs的影像学检查

数字减影血管造影(DSA)是传统意义上诊断脑血管疾病的金标准，结合三维旋转成像可以获得高分辨率的三维血管重建图像，可以清楚显示IVBDAs的位置、动态观察病变血管的血流方式和管腔结构，是介入治疗IVBDAs的基础性检查手段。但DSA同样存在其局限性，如不能显示血管壁和血管腔与周围组织的结构关系，有创性特点也不适用于IVBDAs的动态随访。MRI及CT血管成像具有无创性优势，除显示血管腔外还能同时显示血管壁与血管周围组织，特别是近年来快速发展的高分辨磁共振血管壁成像技术，能够清楚显示夹层动脉瘤的血管壁、壁间血肿、内膜瓣、真腔和假腔[23-25]。结合增强技术还可预测血管内介入治疗后夹层动脉瘤的进展情况[26]。因此，对IVBDAs的诊断和随访来说，多模式影像检查技术的综合应用是未来发展的趋势。

七、IVBDAs的治疗进展

目前，针对IVBDAs的最佳治疗策略尚不明确，仅有的一些小样本观察性研究仅能提供非常低级别的证据。有必要在制定IVBDAs的治疗方案之前先讨论IVBDAs的预期危险分层，危险分层可以参考基础疾病[27]、有无夹层动脉瘤相关的症状体征和症状体征的性质、夹层动脉瘤的位置和形态[28]、夹层动脉瘤的动态变化情况[5]。无症状性IVBDAs和有症状性IVBDAs不能一概而论，导致缺血性卒中的IVBDAs和导致蛛网膜下腔出血的IVBDAs也不能混为一谈。

1.保守治疗：对于无症状性和合并非特异性症状的IVBDAs，单纯随访而不给予特殊治疗措施可能是合理的[8]。对于导致缺血性脑卒中的未破裂IVBDAs，有研究认为包括抗凝和抗血小板治疗在内的抗栓治疗在降低缺血性卒中再发风险的同时并不增加IVBDAs破裂出血的风险[29]，但也有颅内动脉夹层经抗栓治疗后出血的报道[5]。

2.手术治疗：IVBDAs对开颅手术和血管内治疗都是挑战，因为：(1)没有明确的瘤颈；(2)重要的穿支血管经常直接来源于动脉瘤；(3)动脉瘤在颅内的位置常常深在，手术往往是难以到达[30]。外科手术和血管内治疗可以通过减少血流对夹层区域的冲击从而降低IVBDAs的破裂风险并促进夹层修复，已被广泛用于IVBDAs的治疗，包括牺牲载瘤动脉的解构性治疗和保留载瘤动脉的重建性治疗。具体的外科手术方法包括动脉瘤近端闭塞术、动脉瘤孤立术、动脉瘤包裹术、动脉瘤孤立并搭桥术及动脉瘤直接夹闭术[31-32]；动脉瘤包裹术及夹层动脉瘤近端闭塞在相当比例的患者中无效[33-34]；对于必须孤立且累及小脑后下动脉的椎动脉夹层动脉瘤，动脉瘤孤立并搭桥术效果值得期待[35]。与外科手术相比，血管内治疗正方兴未艾，具体方式包括栓塞载瘤动脉、使用单支架[36](包括覆膜支架[37]、血流导向装置[38])或支架套叠[39]、并排支架联合或不联合弹簧圈栓塞技术[40-42]重建载瘤动脉。支架通过对血流的导向及减低夹层假性动脉壁应力并促进内膜形成防止急性期再出血[43]。并行放置支架解决因IVBDAs巨大内腔支架稳定性差及移位的问题。为了进一步增加金属覆盖率用了支架套叠甚至并排支架套叠[39-42]。随着材料科学进步，血流导向装置问世，相比传统的支架，这些支架具有更小的网孔和更大的金属网覆盖面积，既保证正常在血管分支和穿支动脉的灌注，同时大大降低了血流对瘤壁冲击。有学者报道应用血流导向装置治疗IVBDAs[38]，而对于急性出血的IVBDAs，虽有血流导向装置成功治疗的报道[44]，但植入支架需要给予抗凝抗血小板治疗，因而延缓动脉瘤内血栓形成，有可能造成破裂的动脉瘤重复出血[45]。血管内治疗还存在诸如急性或迟发性支架内血栓形成、支架内狭窄、支架辅助栓塞后动脉瘤的长期稳定性此类问题，尚需进一步研究[30]。

综上，IVBDAs的病因及发病机制仍未明确，导致目前的治疗方式无法针对病因进行。近年来有学者提出其与炎症及免疫反应有关，可以考虑使用激素治疗[13]。IVBDAs患者预后取决于临床表现、年龄、病灶形态位置及其毗邻解剖结构等综合情况。临床医生需要对其制定个体化治疗方案最大程度降低手术风险并改善其预后，并不断探索更优的治疗方案。

利益冲突 文章所有作者共同认可文章无相关利益冲突

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