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泌尿科医师实践中的自主反射障碍

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自主性反射障碍 (AVD) 是一种潜在的生命危险状态, 发生在脊椎损伤患者的T6段或更高的水平上。首先, 这种状态表现为无法控制的动脉高血压, 可能导致严重的并发症, 甚至死亡。AVD发展的原因通常是泌尿系统并发症, 以及对下泌尿道的诊断和治疗操作。对于执业泌尿科医生来说, 了解AVD综合征V、其临床表现、诊断和治疗策略以及预防下尿路神经源性功能障碍患者的表现发作是很重要的。AVD被定义为收缩压高于基线20毫米汞柱, 以响应发生在脊髓损伤水平以下的各种传入刺激。AVD基于在位于脊髓T6段上方的节前交感神经元去神经支配和外周 α -肾上腺素能受体过度活跃的情况下, 通过脊髓中的脉冲照射来提高脊髓反射活动。AVD的主要病理生理机制是高去甲肾上腺素血症, 导致位于神经损伤水平以下的皮肤、腹腔和下肢肌肉血管收缩。

关键词: 自主反射障碍; 自主神经反射异常; 神经源性膀胱; 脊髓损伤; 脊髓损伤; 膀胱神经肌肉功能障碍。

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Autonomic dysreflexia in the practice of a urologist

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Autonomic dysreflexia (AD) is a potentially life-threatening condition that develops in patients with spinal cord injury (SCI) at or above the T6 segment. First of all this condition is characterized by uncontrolled arterial hypertension, which can lead to catastrophic complications and even death. The trigger for the development of AD is often urological complications, as well as diagnostic and therapeutic manipulations on the lower urinary tract. It is important for urologists to be aware of the AD syndrome, clinical features of AD, acute and chronic management, as well as prevention episodes of AD in patients with neurogenic lower urinary dysfunction. AD is defined as an increase of systolic blood pressure of 20 mmHg from baseline in response to various afferent stimuli originating below the level of spinal cord injury. AD is based on exaltation of spinal reflex activity with irradiation of impulses in the spinal cord under conditions of denervation preganglionic sympathetic neurons located above the T6 segment and hyperactivity of peripheral α -adrenergic receptors. The main pathophysiological mechanism of AD is hypernoradrenalinemia, leading to vasoconstriction the vessels of the skin, abdominal cavity, muscles below the level of neurological injury.

Keywords: autonomic dysreflexia; neurogenic bladder; spinal cord injury; neuromuscular bladder dysfunction.

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Автономная дисрефлексия в практике уролога

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Автономная дисрефлексия (АвтД) — потенциально жизнеугрожающее состояние, которое развивается у пациентов с позвоночно-спинномозговой травмой на уровне или выше сегмента Т6. Прежде всего это состояние проявляется неконтролируемой артериальной гипертензией, которая может привести к тяжелым осложнениям и даже летальному исходу. Причиной развития АвтД чаще всего становятся урологические осложнения, а также диагностические и лечебные манипуляции на нижних мочевыводящих путях. Практикующим урологам важно быть осведомленными о синдроме АвтД, его клинических проявлениях, диагностической и лечебной тактике, а также профилактике предотвращения эпизодов ее манифестации у пациентов с нейрогенной дисфункцией нижних мочевыводящих путей. АвтД определяется как повышение систолического артериального давления выше базового значения на 20 мм рт. ст. в ответ на различные афферентные стимулы, возникающие ниже уровня повреждения спинного мозга. В основе АвтД лежит экзальтация спинальной рефлекторной активности с иррадиацией импульсов в спинном мозге в условиях деннервации преганглионарных симпатических нейронов, расположенных выше сегмента Т6 спинного мозга и гиперактивности периферических α -адренорецепторов. Основной патофизиологический механизм АвтД — это гипернорадреналинемия, приводящая к вазоконстрикции сосудов кожи, брюшной полости, мышц нижних конечностей, расположенных ниже неврологического уровня повреждения.

Ключевые слова: автономная дисрефлексия; вегетативная дисрефлексия; нейрогенный мочевой пузырь; травма спинного мозга; позвоночно-спинномозговая травма; нервно-мышечная дисфункция мочевого пузыря.

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在脊椎损伤 (SCI) 的远端后果中, 以及运动和感觉障碍, 植物和心血管并发症的综合表现为对这些系统失去足够的中央控制[1, 2]。这些并发症包括自主反射障碍 (AVTD)。它发生在SCI位于或高于脊髓(SM)的第六胸椎(T6)节段时。AVD的特点是自主神经系统反应过度, 血压显著升高以响应SM损伤水平以下的刺激。这种刺激物最常与膀胱或直肠的显著溢出有关。在长期感觉障碍的背景下, AVD可能是泌尿系统功能不全的唯一症状, 从膀胱过度拥挤到肾绞痛引起的疼痛[3]。

定义

自主性反射障碍 (AVD) 是生长神经系统交感系的一种增强反应, 在T6段或以上的CM病变患者中, 由于刺激性冲动, 表现为超去甲肾上腺素发作, 低于神经损伤水平。在欧洲泌尿外科协会的指南中AVD被定义为收缩压从基线突然升高 >20 毫米汞柱[4, 5]。

分布量

在T6段及以上接受SCI的神经源性排尿障碍患者中, 48-90%会出现自主神经反射异常[6]。AVD 最常见于颈椎损伤, 发生在超过70%的C1-C8段损伤患者中。同时, 在T1-6段级别的SM损伤情况下AVD的发生频率约为30%。

通常, AVD症状会在6-12周内出现。创伤后, 由于在早期阶段, 一种对血流动力学障碍(脊髓休克期)的补偿是可能的, 因此在长期SCI期间AVD的患病率增加。研究发现38.7%的患者在SCI后的前2年内发生AVD。同时, 如果损伤持续时间超过2年, 80%的患者出现AVD[7]。

发病机理

SCI期间发生的SM损伤, 位于心脏交感中枢所在的Th6节段上方或水平, 剥夺了节前神经元的脊髓上集中影响, 导致去神经反射亢进, 伴有高去甲肾上腺素血症[1, 8]。由于SCI中断下行和上行神经通路, 位于损伤水平以下的节前神经元保留与脊髓内神经元的通讯, 并可通过损伤后未受损的脊髓反射通路激活。当内脏或躯体纤维受到低于神经损伤水平的刺激时, 节前心脏神经元兴奋。分散性SM条件下, 大脑皮层和脑桥泌尿中枢缺乏抑制性反应, 导致逼尿肌-括约肌协同失调, 增强脊髓反射, 导致AVD。

AVD最常见的触发因素是膀胱壁过度扩张并明显溢出、直肠溢出、对泌尿道的侵入性干预(尿动力学检查、膀胱镜检查、膀胱导管插入术等)、疼痛刺激(受伤、褥疮、紧身衣)。性刺激、射精、尿路感染表现、膀胱结石、直肠检查和远程冲击波碎石术也可能是AVD的原因[9]。大多数作者认为在日常生活中AVD最常见的诱发因素是膀胱溢出(89%的患者)和疼痛刺激(75%的患者)[10]。

临床表现

AVD的临床表现可以用一系列症状来描述: 突然和持续的动脉高压(>20 毫米汞柱从初始基线), 不同强度的头痛, 瞳孔扩张、寒战、皮损伴斑点性充血和职业性出汗高于脊髓损伤、呼吸困

难, 增强骨骼肌的自发痉挛, 以及脉搏频率的变化[4]。根据R. Solinsky和合著者的数据, AVD的临床表现研究[11]在AVD发作期间68%的患者出现心动过速, 心率平均每分钟增加20次, 虽然心动过缓的发生率要低得多, 占有记录病例的0.3%。

AVD突然出现, 如果不及时治疗, 可能会产生极其严重的后果: 蛛网膜下腔出血, 高血压性脑病, 视网膜脱离, 心律失常, 痉挛, 神经源性肺水肿, 肾衰竭(由于长期的肾血管收缩), 死亡。

诊断

尽管AVD在SMI患者中很常见, 但并非所有可能遇到这种情况的专家都能及时识别。AVD的诊断标准是收缩压突然升高 >20 毫米水银柱从基线和上述临床表现来看。在这种情况下, 必须考虑到T6段以上SM损伤水平高的患者的特点是动脉低血压。通常, 他们的基线收缩压比一般人群低15-20毫米水银柱[9]。

治疗和预防

在发生AVD的情况下, 观察明确的治疗措施算法非常重要。识别和消除引起AVD的外源性/内源性因素似乎是治疗和预防这种疾病的关键, 连续监测血流动力学参数(每5分钟)可以评估所采取措施的有效性[13]。

由于AVD最常见的原因是膀胱过度拥挤, 应立即排空膀胱, 停止下尿路的侵入性操作。内脏刺激的第二个最常见原因是直肠溢出。为了消除这一因素, 必须疏散直肠安甬的内容物。结论利多卡因在诊断过程中对肛门区域的灌注有助于降低血压升高[16]。如果AVD的原因不明显, 应进行彻底检查, 以排除褥疮、烧伤、皮肤感染、不同部位的脓肿、急性腹部(阑尾炎、急性肠梗阻、腹膜炎等)。

患者应坐在椅子上或抬起床的头端, 以产生一种有助于血压自然下降的正压效应。

如果上述所有操作都无效, 建议改用药理矫正方法[13, 14]。

迄今为止, 还没有普遍接受的治疗AVD期间高血压危险的药理学方案; 文献中描述了解决这种情况的各种方法。大多数作者指出在AVD发作期间使用10毫克硝苯地平可更有效地实现快速降压效果[15, 17, 18]。介绍了血管紧张素转化酶抑制剂(ACEI)与钙通道二氢吡啶阻滞剂的成功同时应用。在这种情况下经舌下服用25毫克卡托普利及其在30分钟内降低血压的疗效不足观察, 患者给予5毫克尼非地平[19]。在专门的文献中, 提到了一个成功的静脉输液双氢吡啶钙通道阻滞剂尼卡地平用于购买AVD的临床案例[20]。

AVD的预防旨在减少发作频率及其扩展的临床表现。众所周知由于存在发生AVD的风险, 对颈椎和上胸椎受损的CM患者进行逼尿肌肉毒素注射的经验仍然相对有限, 已有数据表明, 逼尿肌肉毒素杆菌病后AVD发作的发生率显著降低。根据I. Y. Jung和合著者[23]将肉毒杆菌毒素注射到对CM高度损伤的患者的膀胱壁中, 可以降低收缩压的平均值。持续使用抗胆碱能药物是预防下尿路神经源性功能障碍引起AVD的一种有希望的方法。目前, 正在进行一项研究, 以评估选择性M-抗胆碱能拮抗剂(非索替罗定)对自身

免疫性疾病表现的频率和严重程度的影响[22]。众所周知, SMI患者持续服用 α -1受体阻滞剂会降低AVD发作的发生率, 但是由于动脉低血压恶化的风险, 许多研究人员对此存在争议。在进行尿动力学研究前30分钟服用硝苯地平可有效预防颈椎损伤患者出现AVD症状。众所周知, 持续摄入钙通道阻滞剂会导致动脉或直立性低血压增加[21]。

因此, AVD可被归类为SMI表现的危险并发症。AVD可以描述为高SCI水平患者对各种疼痛刺激和下尿路不适, 特别是膀胱过度溢出的不可控交感反应。AutoD状态被解释为收缩压突然升高 >20 毫米水银柱从基线开始。治疗应分为非药物治疗和药物治疗, 缓解急性

症状, 以及预防措施。预防AVD发展的算法尚未最终开发出来, 但是在有希望的方法中, 可以选择持续摄入M-抗胆碱药和向逼尿肌注射肉毒杆菌毒素。

最重要的是告知AVD发展的可能原因、其临床表现及其预防, 不仅对康复科医生、泌尿科医生和全科医生, 也对患者本人和SMI患者的护理人员。

附加信息

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