

个案报道

破裂型颈椎间盘突出症患者脱出髓核组织自发吸收1例报告

Resorption of herniated cervical nucleus pulposus: a case report

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突出椎间盘髓核组织的自发吸收(resorption of herniated nucleus pulposus,RHNP)是指椎间盘突出症患者未经手术切除突出的髓核组织或进行髓核消融等干预措施而发生的髓核缩小或消失的现象^[1]。近年来,随着MRI检查的普及,越来越多的报道发现椎间盘突出后存在自发吸收现象,尤以腰椎间盘突出相关报道较多^[2-3],而颈椎间盘突出组织自发吸收现象国内外报道较少^[4-8]。2016年3月我科门诊随访1例破裂型颈椎间盘突出症患者,6个月后患者椎间盘突出组织部分自发吸收,不适症状显著改善,报告如下。

患者女,51岁,无明显诱因出现双上肢疼痛、无力1个月,加重3d,偶有“踩棉感”,于2016年3月20日来我

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院门诊就诊。查体:颈椎前屈、后伸活动受限,C4~C6棘突压痛,椎间孔挤压试验(+);左上肢桡侧皮肤及左手拇指浅感觉减退,左侧肱二头肌、伸腕肌、肱三头肌肌力4级;双侧Hoffmann征(+).颈椎MRI检查示多个椎间盘含水量下降,C5/6椎间盘中央偏左侧突出,颈脊髓受压,左侧神经根明显受压(图1)。结合患者症状、体征及影像学资料,诊断为破裂型颈椎间盘突出症。患者拒绝手术治疗,拒绝口服营养神经及镇痛类药物,仅间断性外用膏药(奇正消痛贴)缓解症状,并改善生活习惯,如避免长时间低头等。6个月后,患者自觉双上肢疼痛明显缓解,双上肢力量增加,“踩棉感”消失。门诊查体:颈椎前屈、后伸及旋转活动度正常,颈椎各棘突无压痛及叩击痛,椎间孔挤压试验(-);双上肢皮肤浅感觉未见异常,双上肢肌力均为5级;双侧Hoffmann征(+).复查颈椎MRI示C5/6椎间盘突出范围较前明显缩小,颈脊髓轻度受压,左侧神经根轻度受压(图2)。1年后患者双上肢疼痛、无力消失,仅有间断性颈部酸

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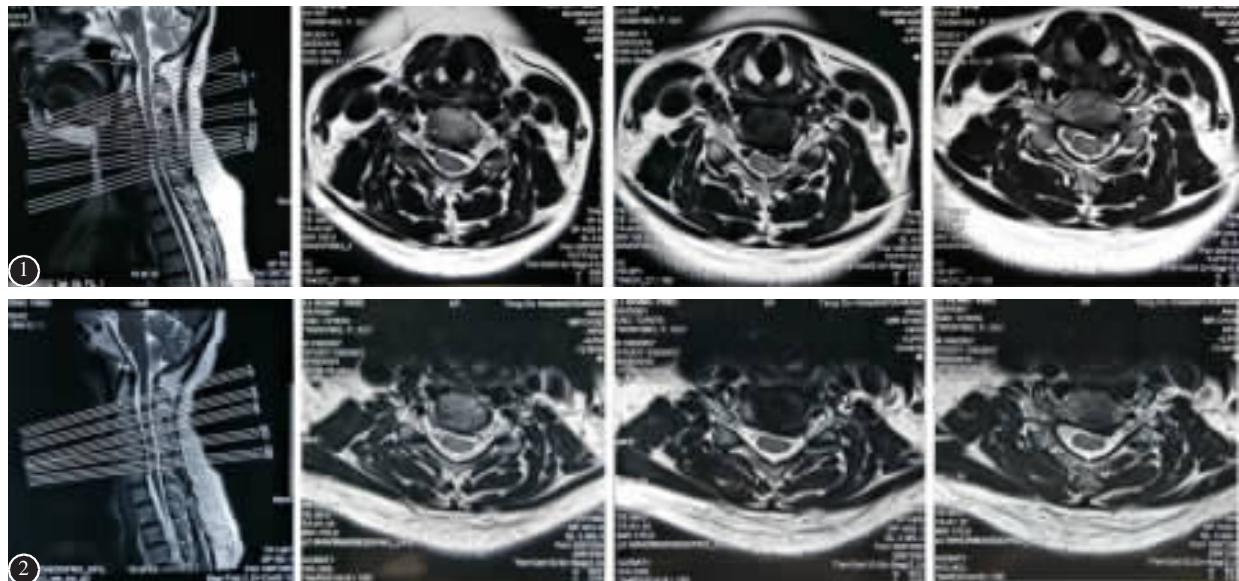


图 1 患者首次就诊颈椎 MRI 检查:矢状位扫描可见颈椎生理曲度消失,多个椎间盘含水量下降,椎间盘突;C5/6 椎间盘轴位扫描可见 C5/6 椎间盘中央左侧突出,颈脊髓受压,左侧神经根明显受压 **图 2** 6 个月后复查颈椎 MRI:矢状位扫描可见颈椎间盘突出程度较前减轻;C5/6 椎间盘轴位扫描可见 C5/6 椎间盘突出较首次就诊时显著缩小,颈脊髓受压及左侧神经根受压程度较前明显减轻

Figure 1 The MRI examination of first visit: Cervical intervertebral disc MRI on sagittal scans showed abnormal cervical curvature, Low water content in cervical disc and herniated cervical disc; Axial MRI images showed the C5/6 intervertebral disc herniated at the left of center, the spinal cord and nerve root was compressed obviously **Figure 2** The MRI examination, six months after first visit: Cervical intervertebral disc MRI on sagittal scans showed the herniated cervical disc was smaller compared with first visit; Axial MRI images showed the C5/6 intervertebral disc herniated at the left of center, the severity of compression on spinal cord and nerve root was significantly relieved compared with first visit

困惑,未行 MRI 检查。

讨论 破裂型椎间盘突出症是指突出的髓核组织突破后纵韧带,直接压迫硬膜囊或神经根,由于颈椎管空间小,突出组织往往造成严重的神经症状,其中有少数患者在随后的复查中发现脱出的椎间盘组织出现不同程度的吸收,并伴随症状缓解。

腰椎间盘突出后自发吸收现象最早由 Guinto 等^[9]于 1984 年首次报道,此后国内外相继有相关报道。1992 年 Krieger 和 Maniker 等^[10]报道了颈椎间盘突出组织自发吸收的病例,但目前颈椎间盘突出后自发吸收的相关报道仍然较少,其具体的发生机制目前还未阐明。

椎间盘是一个复合结构体,由覆盖于上下的骨性终板、软骨性终板,环绕于外层的纤维环和中心的髓核共同组成。正常的椎间盘是一个无血管组织,其营养交换主要通过终板的弥散功能完成^[11]。从免疫学角度看,椎间盘是人体内最大的无血管组织,其中的髓核外包绕着纤维环,椎间盘自出生后就与外周血液循环隔绝,因而具备自身抗原性。髓核的抗原成分在突破后纵韧带后会暴露到循环系统中,这可能会引发一系列的免疫反应^[12]。目前研究发现,破裂型椎间盘突出是最常见的自发吸收类型,后纵韧带完整与否是决定能否自发吸收或缩小的关键因素之一^[13]。

现阶段研究认为,突出椎间盘组织的自发吸收与椎

间盘免疫赦免平衡的打破^[12]、炎性反应^[14,15]、突出组织的脱水作用^[16]、血管化^[17]、突出组织降解与细胞凋亡^[11,17]等机制密切相关。Murai 等^[18]的研究发现腰椎间盘髓核突出后纵韧带后与硬膜外腔血管环境接触从而引发了自身免疫反应,其中自然杀伤细胞(NK 细胞)和巨噬细胞均参与了早期的自身免疫反应;我们也通过实验研究证实了类似的机制^[12]。在炎性反应机制研究方向上,Tsuru 等^[19]通过电镜发现大量的巨噬细胞浸润至突出的椎间盘组织中,认为此机制为浸润的巨噬细胞吞噬椎间盘组织碎片所造成。Kobayashi 等^[5]的研究进一步证实巨噬细胞分泌的溶酶体酶在细胞外同时发挥着吞噬作用,同时体内的多种炎性介质也参与其中。Rätsep 等^[17]也发现脱出的髓核组织与局部血管化关系比未破裂型更为密切,这提示新生血管化在髓核组织自发吸收中发挥了重要作用。近年来,越来越多的研究从不同角度阐释了脱出椎间盘组织自发吸收的机制。为此,北美脊柱外科学会 24 位专家于 2014 年在 The Spine Journal 上联合发表了《An evidence-based clinical guideline for the diagnosis and treatment of lumbar disc herniation with radiculopathy》一文,指出腰椎间盘突出部分可发生缩小或消退,众多研究显示随着椎间盘突出程度的减轻,患者临床症状得到改善^[20]。

本例患者发病无明显诱因,且病程短,患者颈椎 MRI

可见多个椎间盘含水量下降,生理曲度丧失,存在颈椎退变的基础。而患者就诊时发病1个月,可能存在颈椎间盘在退变的基础上发生C5/6椎间盘急性突出,从而出现病情加重3d;在此基础上局部炎症介质释放,免疫平衡打破,导致相邻神经根的刺激症状,如左侧肱二头肌肌力减弱;且短时间内出现的压迫导致椎管空间较之前变小,从而出现双侧症状。本患者首次就诊时,根据MRI结果患者属于旁中央型颈椎间盘突出症,因此,患者同时存在根性症状及脊髓受压症状,经外用膏药及改善生活习惯6个月后,患者复查MRI可见突出髓核组织部分吸收,颈脊髓及神经根受压程度明显缓解,症状显著改善。

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