

· 综述 ·

粘连性肩关节囊炎: 发病机制、诊断及治疗的研究进展

王 瑀, 曾令清, 潘寅兵*

南京医科大学第一附属医院麻醉与围术期医学科, 江苏 南京 210029

[摘要] 粘连性肩关节囊炎是导致肩部疼痛的常见原因, 主要表现为肩关节疼痛、活动受限和功能障碍。目前, 该病的病因及发病机制尚未明确, 临床诊断主要依赖病史、体格检查和影像学资料。国内外治疗方法有多种, 尚未达成共识。文章将结合最新研究成果, 综述粘连性肩关节囊炎的发病机制、诊断及治疗的进展。

[关键词] 粘连性肩关节囊炎; 发病机制; 诊断; 治疗

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Adhesive capsulitis of the shoulder: research progress in pathogenesis, diagnosis and treatment

WANG Yu, ZENG Lingqing, PAN Yinbing*

Department of Anesthesiology and Perioperative Medicine, the First Affiliated Hospital of Nanjing Medical University, Nanjing 210029, China

[Abstract] Adhesive capsulitis of the shoulder is a prevalent cause of shoulder pain, characterized by pain, limited range of motion and functional impairment. Currently, the etiology and pathogenesis of the disease remain unclear, with clinical diagnosis primarily based on medical history, physical examination and imaging data. There are various treatment approaches available both domestically and internationally, but consensus has yet to be reached. This article will review the latest research on the pathogenesis, diagnosis, and treatment advancements of adhesive capsulitis of the shoulder.

[Key words] adhesive capsulitis of the shoulder; pathogenesis; diagnosis; treatment

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粘连性肩关节囊炎, 又称冻结肩、肩周炎或五十肩, 是一种常见的肩部疾病, 好发于 50 岁左右的中老年人。其主要表现为肩关节疼痛及主动、被动活动受限。该病分为原发性和继发性两类, 前者病因不明, 亦称为特发性粘连性肩关节囊炎, 是指无明确肩部外伤或疾病史的自发性关节僵硬; 后者则有明确病因, 通常继发于外伤、手术或其他肩部疾病^[1]。

粘连性肩关节囊炎是一种自限性疾病, 在缺乏干预的情况下, 1~2 年内多数可逐渐缓解, 对于疼痛

和活动受限较重的患者, 往往症状缓解欠佳^[2]。有研究显示, 接受保守治疗的患者中, 20%~40% 仍存在持续性疼痛和功能受限^[3]。因此, 积极有效的早期干预对于缓解疼痛和恢复肩关节功能至关重要。目前, 该病的治疗方法较多, 不同治疗方法之间的疗效差异较大。文章旨在对粘连性肩关节囊炎的发病机制、诊断及治疗的近期研究进展进行综述, 以期临床诊疗提供参考。

1 流行病学

在正常人群中, 粘连性肩关节囊炎的发病率为 2%~5%, 且好发于 40~60 岁的中老年人, 女性发病率较高^[4]。有研究指出, 非优势侧的发病率高于优

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*通信作者 (Corresponding author), E-mail: panyinbing@sina.com (ORCID: 0000-0003-4102-2962)

势侧(58.9% vs. 41.1%)^[5]。与粘连性肩关节囊炎相关的危险因素包括糖尿病、甲状腺疾病、帕金森病、自身免疫性疾病和吸烟等。其中, 糖尿病被视为该病的主要危险因素, 45岁以上糖尿病患者的发病率可达59%, 且活动受限程度和致残风险更高^[6]。

2 发病机制

肩关节囊薄而松弛, 包裹肩关节的纤维鞘主要由致密的 I 型胶原蛋白和弹性纤维束构成, 且血管和神经数量有限。在粘连性肩关节囊炎患者中, 典型的胶原结构遭到破坏, 导致结缔组织纤维化及邻近滑膜增厚。这些纤维化改变伴随炎症、新生血管和神经形成, 进而引起关节腔减小和关节囊僵硬, 最终导致疼痛和活动受限^[7]。目前该病发病机制仍不确定, 可能涉及炎症反应、纤维化、血管及神经变化、代谢变化等(图1)。

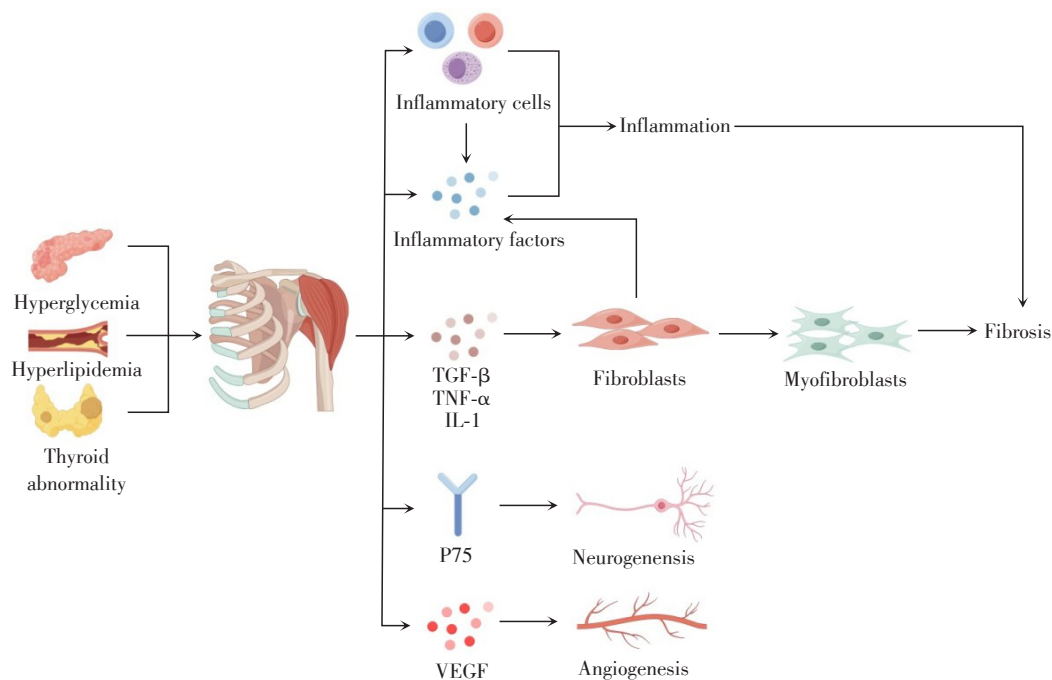
2.1 炎症反应

粘连性肩关节囊炎患者的肩关节囊及周围软组织存在广泛的无菌性炎症, 这与毛细血管再生增加、成纤维细胞增殖、滑膜增厚及细胞外基质沉积增加相关^[8]。在病变的滑膜组织中, 存在多种炎症

细胞, 包括B细胞、T细胞、巨噬细胞和肥大细胞。炎症介质如白细胞介素(interleukin, IL)-1、IL-6、IL-10及肿瘤坏死因子- α (tumor necrosis factor α , TNF- α)等在病变滑膜组织中表达失衡, 并参与驱动炎症反应^[9]。研究发现, 与健康成纤维细胞相比, 病变组织中的成纤维细胞产生更高水平的促炎细胞因子, 如IL-6、IL-8和趋化因子配体20^[10]。

2.2 纤维化

纤维化是粘连性肩关节囊炎的基本病理过程。许多研究表明纤维化与转化生长因子- β (transforming growth factor- β , TGF- β)密切相关, 其在病变组织中高度表达, 并可诱导多种纤维化反应, 包括成纤维细胞增殖和肌成纤维细胞分化等^[11]。成纤维细胞是关节囊内的主要细胞类型, 负责产生细胞外基质蛋白, 参与组织结构的形成。TGF- β 可诱导成纤维细胞转化为肌成纤维细胞, 后者是纤维化反应的关键效应细胞。此外, 在正常动态平衡下, I 型胶原蛋白是主要的基质蛋白, 起到支撑和保护的作用; 而在病理条件下, 细胞外基质周转加快, III型胶原数目增加, 导致关节囊稳定性降低^[12]。



Under the influences of hyperglycemia, hyperlipidemia, thyroid dysfunction, and other factors, inflammatory cells and cytokines become dysregulated within affected tissues, driving inflammatory responses. TGF- β induces the transformation of fibroblasts into myofibroblasts, which are key effector cells in fibrosis. Concurrently, fibroblasts in the affected tissue produce higher levels of pro-inflammatory cytokines. Increased vascularization is another major manifestation in the disease process, associated with the overexpression of VEGF. Additionally, neovascularization is often accompanied by nerve formation, which may be related to the increased expression of the p75 neurotrophin receptor.

图1 粘连性肩关节囊炎病理生理机制

Figure 1 The pathophysiological mechanisms of adhesive capsulitis of the shoulder

2.3 血管及神经变化

在粘连性肩关节囊炎的发病进程中,血管再生可能发挥关键作用。关节镜下常见滑囊血管炎和血管肉芽组织形成,这与血管再生相关。血管内皮生长因子(vascular endothelial growth factor, VEGF)是控制该过程的主要生长因子,作为一种结合糖蛋白,其能够增强血管通透性,促进血管内皮细胞的增殖和迁移,最终导致新生血管的形成。新生血管形成的同时常伴随新生神经形成,这可能与神经生长因子受体p75表达增加有关^[10]。

2.4 代谢变化

糖尿病与粘连性肩关节囊炎之间存在显著相关性^[13],尤其是在长期血糖控制不佳的情况下,究其原因可能是高血糖放大了促炎和促纤维化的信号级联反应。研究发现糖尿病患者体内持续升高的促炎因子,如TNF- α 、IL-6和IL-1 β ,在关节囊及滑膜中也维持在较高水平^[14]。此外,晚期糖基化终末产物(advanced glycation end product, AGE)通过多种机制促进糖尿病患者体内器官的纤维化和炎症反应:一方面,AGE在胶原蛋白分子之间形成共价交联,引起胶原纤维超微结构改变,提高其对蛋白水解作用的抗性,导致胶原硬度增加、组织顺应性降低^[15-16];另一方面,AGE与其受体RAGE(the receptor of advanced glycation end product)结合,通过核因子- κ B(nuclear factor- κ B, NF- κ B)途径促进成纤维细胞黏附分子-1(intercellular cell adhesion molecule-1, ICAM-1)和IL-6的表达,刺激基质细胞和免疫细胞产生促炎因子,加重炎症反应,进一步促进该病的病理进程^[17]。

血脂升高也与粘连性肩关节囊炎的发生和发展密切相关。脂蛋白增高与血管炎症和免疫反应相关,是该病的独立危险因素。在早期患者中,血脂水平与肩关节功能评分呈负相关,显示出两者的相关性^[18]。研究发现,非高密度脂蛋白胆固醇血症与该病显著相关,其中载脂蛋白A1的表达水平可反映病情的严重程度^[19]。

此外,甲状腺功能障碍也与粘连性肩关节囊炎发病风险增加有关,尤其是甲状腺功能减退症。甲状腺滤泡旁细胞分泌的降钙素可能是连接甲状腺功能障碍与该病的桥梁。研究发现,粘连性肩关节囊炎患者接受鲑鱼降钙素治疗后,症状可改善^[20]。

3 分期及诊断

Neviaser^[21]在1945年描述了粘连性肩关节囊炎

的4个临床阶段。Ⅰ期:疼痛期(0~3个月),主要表现为肩部疼痛,以夜间疼痛为主,影响睡眠,而活动度受限不明显。Ⅱ期:渐冻期(3~9个月),临床症状以剧烈疼痛为主,同时伴随肩部各个方向主动和被动活动度的逐渐降低。Ⅲ期:冻结期或粘连期(9~15个月),主要表现为肩部活动明显受限,而疼痛则有所减轻。Ⅳ期:解冻期或缓解期(15~24个月),其特点是疼痛逐渐减轻,肩关节活动度逐渐改善。

目前,粘连性肩关节囊炎的诊断标准尚未统一。实验室检查多无异常,其临床诊断主要依据详细的病史、体格检查和影像学资料。患者多无外伤史,早期在无明显诱因的情况下出现肩部疼痛,活动时疼痛加重,且夜间疼痛较为严重;同时伴有肩部活动受限,以肩关节外展、外旋和后伸受限最为明显。体格检查中,部分患者可见三角肌轻度萎缩,冈上肌腱、冈下肌腱及肩胛下肌腱等部位可有明显压痛,搭肩试验和摸耳试验可能呈阳性反应。

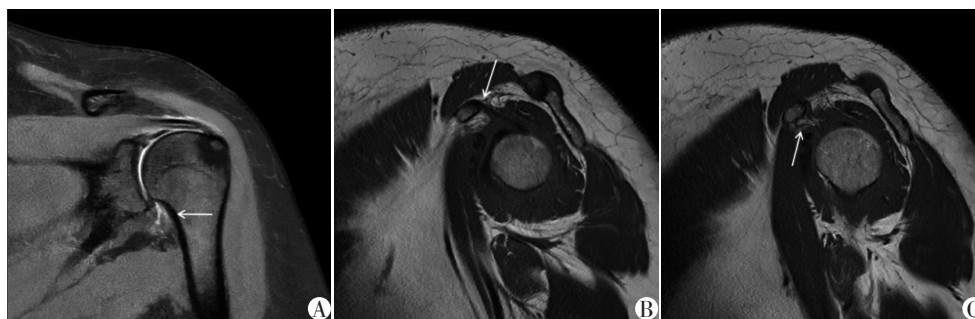
临床诊断常采用X线、MRI、超声等影像学检查。X线检查多无明显异常,主要用于排除骨折、脱位、钙化性肌腱炎等疾病。MRI能清晰显示病变的具体部位及严重程度,在粘连性肩关节囊炎的诊断中具有重要价值(图2)。其中,喙肱韧带和肩袖间隙具有高度特异性,尤其是喙肱韧带厚度 ≥ 3 mm时准确性最高^[22]。最近的研究表明,腋窝关节囊厚度 > 4.5 mm、肩袖间隙厚度 > 6 mm时,其诊断灵敏度分别为91%和88%,特异度均为90%,准确度分别为90%和89%^[23]。MRI在鉴别肩袖损伤、肩部肿瘤等疾病中也展现出明显优势。超声具有易获得、成本低、快捷方便的优势,能够动态评估临床症状与影像学表现之间的相关性^[24-25],并可在超声引导下进行局部注射治疗。

4 治疗

粘连性肩关节囊炎的治疗目标是减轻患者疼痛,改善关节功能。该病的发病机制尚不完全明确,需要根据患者的病史、症状、体征及疾病分期选择合适的治疗方案^[26]。目前治疗方法尚未形成共识,对于早期患者,推荐首选保守治疗^[27]。若疗效不佳或病程较长,可考虑侵入性治疗或多种治疗方法联合的方案(表1)。

4.1 健康教育

良好的健康教育是重要的干预措施之一。让患者了解该病的发病因素、临床表现、治疗方案、转归和预后,有助于消除患者的焦虑,提高治疗依从



The case is a 43-year-old female presenting with over 6 months of left shoulder pain. A: Coronal FS T2-weighted imaging(T2WI), showing thickening of the axillary joint capsule (indicated by the white arrow). B: Sagittal T2WI, showing thickening of the coracohumeral ligament (indicated by the white arrow). C: Sagittal T2WI, revealing the disappearance of the subcoracoid fat triangle (indicated by the white arrow) and narrowing of the rotator cuff interval.

图2 粘连性肩关节囊炎 MRI 表现

Figure 2 The MRI manifestations of adhesive capsulitis of the shoulder

性,同时使患者认识到当前治疗方法的多样性和局限性。

4.2 非手术治疗

4.2.1 口服药物

非甾体类抗炎药(non-steroidal anti-inflammatory drug, NSAID)是常用的口服药物,可用于消炎镇痛,但对关节活动度没有明显改善作用。炎症和纤维化是该病发病机制中的两个重要方面,因此,在早期炎症阶段使用NSAID更为有效。口服糖皮质激素能够在短期内明显缓解疼痛并改善活动度和功能,其效果可持续约6周^[28]。此外,可根据需要口服盐酸乙哌立松等中枢性肌松药,以改善肌肉紧张状态。

4.2.2 物理治疗

物理治疗作为一种常用的干预措施,主要用于改善并维持关节活动度,通常与其他治疗方法联合使用^[29]。根据疾病的不同阶段,常采用多种治疗方法,包括钟摆运动、被动牵拉、冲击波、激光和脉冲电磁场等疗法^[30-32]。

体外冲击波疗法(extracorporeal shock wave therapy, ESWT)是一种非侵入性治疗手段,利用体外治疗仪产生冲击波能量,通过探头将能量聚焦于治疗部位。该疗法具有松解粘连,改善局部血供以及促进炎症和水肿消散的作用。作为一种物理治疗方法,ESWT无药物相关不良反应,操作简单、并发症少、安全性高,已成为一种重要的非手术治疗方法^[33-34]。

本体感觉神经肌肉促进疗法(proprioceptive neuromuscular facilitation, PNF)是一种通过刺激本体感受器以促进相关神经肌肉反应的治疗技术,旨

在提高肌力、耐力和协调性,扩大关节活动范围,缓解肌肉紧张,改善关节功能^[35]。研究发现,额外的本体感觉训练能够更有效地改善疼痛和功能活动^[36]。

4.2.3 注射治疗

关节内注射糖皮质激素可在该病的早期阶段进行,相较于口服该类药物,局部注射药物浓度更高,能够在短期内更快速有效地减轻炎症、缓解疼痛并改善活动度。推荐在肩袖间隙、孟肱关节和肩峰下滑囊进行联合注射^[37],该方法疗效显著、安全可靠且复发率低。关于注射剂量,研究表明低剂量(20 mg)与高剂量(40 mg)的曲安奈德均能显著改善患者的疼痛和活动度评分,且两者之间差异无统计学意义^[38]。因此,建议在早期首选低剂量激素注射,特别是对合并糖尿病的患者,以减少血糖波动。

玻璃酸钠是关节滑液的主要成分,具有润滑关节、营养软骨的作用。局部注射玻璃酸钠可减少关节摩擦,起到缓解疼痛、减轻粘连的作用。研究表明,玻璃酸钠能够抑制成纤维细胞的增殖及Ⅲ型前胶原蛋白mRNA的表达,且呈剂量依赖性,这有助于减轻患肩的粘连及纤维化^[39]。

富血小板血浆(platelet rich plasma, PRP)是从自体血中制备的高浓度血小板血浆,富含多种生长因子和细胞因子,具有促进细胞增生、组织修复以及消炎镇痛的作用。一项研究比较了接受PRP注射治疗与接受糖皮质激素注射治疗的患者,结果显示在6个月的随访中,PRP组在疼痛缓解、肌肉力量和肩部功能改善方面达到了与激素组相当的水平,且PRP组的所有改善均维持更长时间^[40]。

4.2.4 关节囊扩张术

该方法通过向关节囊内缓慢注入足量液体,如

无菌生理盐水、糖皮质激素、局部麻醉药等,利用液体压力使关节囊膨胀、扩张,直至关节囊内的粘连组织松解,从而达到缓解疼痛、改善活动范围的目的,此方法常用于冻结期^[41]。

4.2.5 神经阻滞

肩胛上神经支配约70%的肩部感觉,肩胛上神经阻滞通常用于治疗术后或创伤所致的难治性肩痛,也可用于粘连性肩关节囊炎的治疗。研究发现,肩胛上神经阻滞可在短期内显著缓解患者疼痛,但功能改善通常较晚出现^[42]。

4.2.6 其他

降钙素能够减轻炎症反应并刺激内啡肽释放。研究证实,鲑鱼降钙素可以改善与纤维化相关分子的mRNA表达^[43]。一项随机双盲对照试验发现,鼻喷降钙素与安慰剂治疗相比,6周后降钙素组患者的肩痛、关节活动度和功能评分显著改善^[44]。

4.3 手术治疗

4.3.1 肩胛上神经脉冲射频术

脉冲射频是近年来用于治疗慢性疼痛疾病的一项微创介入技术。研究发现,与接受假治疗的对照组相比,肩胛上神经脉冲射频在术后第6、12周时的疼痛评分显著降低,且无严重不良反应^[45]。

4.3.2 麻醉下手法松解术(manipulation under anesthesia, MUA)

MUA是在局部麻醉或全身麻醉下,通过专业手法拉伸纤维化的关节囊,从而解除粘连,短期内可获显著的活动度改善。该手术通常在臂丛麻醉下进行,是治疗粘连性肩关节囊炎的常用方法^[46]。麻醉后,术者对肩关节进行各个方向的被动伸展,包括前屈、外展、外旋、内旋和后伸等方向,术中常可听到关节囊撕裂的“咔咔”声或棉帛样撕裂声。关

于实施MUA的最佳时间尚未形成共识,建议对非手术治疗6个月无效的患者考虑进行MUA。

4.3.3 关节镜下关节囊松解术(arthroscopic capsular release, ACR)

ACR是在全身麻醉下进行的微创手术,由于在关节镜直视下进行手术,其对病变部位的松解更为彻底且精准,减少了并发症的出现。多项研究已证实ACR的有效性,可显著降低疼痛评分,并改善肩部功能和活动度^[47-48]。在最近的一项多中心三臂随机对照试验发现,物理治疗、MUA和ACR在治疗粘连性肩关节囊炎的疗效方面并不优于彼此,且ACR成本更高,而MUA是成本效益最优的选择^[49-50]。

4.3.4 开放手术

近年来,随着关节镜技术的广泛应用,开放手术在粘连性肩关节囊炎治疗中的应用逐渐减少,已不再作为该病的首选治疗方法。

5 总结与展望

粘连性肩关节囊炎作为临床常见病,患病人群较多。该病具有一定的自限性,若不积极加以干预,可能导致病程延长和功能障碍,严重时影响生活质量。对于该病的诊断标准和最佳治疗方法目前尚未形成共识,推荐根据疾病不同分期制定个体化治疗方案。随着对该病危险因素和发病机制的深入研究,将会有越来越多有效的治疗方法用于临床。未来的研究方向应集中于开展高质量的临床研究,比较各种治疗方法的疗效差异,并深入探索疾病机制,发现新的治疗靶点,以提高治疗有效率。

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表1 原发性冻结肩不同阶段的治疗措施

Table 1 Treatments in different stages of adhesive capsulitis of the shoulder

Stage	Duration(months)	Pharmacotherapy	Interventional treatment	Surgical intervention
I, Pain	0-3	NSAIDs Corticosteroid	Intra-articular injection	-
II, Freezing	3-9	NSAIDs	Hydrodistension Intra-articular injection suprascapular nerve block Suprascapular nerve pulsed radiofrequency	-
III, Frozen	9-15	NSAIDs	Hydrodistension Intra-articular injection PRP	MUA ACR
IV, Thawing	15-24	-	Intra-articular injection PRP	MUA ACR

MUA: manipulation under anesthesia; ACR: arthroscopic capsular release.

京医科大学, 但不存在任何可能影响本研究成果客观性的利益冲突。论文的撰写评审与发表决策均保持独立性与学术诚信。

Conflict of Interests:

All authors declare that although all authors are currently affiliated with Nanjing Medical University, there are no personal relationships that could be perceived as influencing the objectivity of this research. The research manuscript preparation, peer review, and publication decisions were conducted independently and with academic integrity.

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王瑀负责文献收集、文献阅读、文献核对、论文初稿撰写; 曾令清参与文献核对、论文初稿撰写; 潘寅兵提出研究方法、文献核对、论文审阅与修改。

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