

NGH分类辅助下的夏科氏肩关节病影像学评估与治疗

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[摘要] 夏科氏肩关节病是一种罕见的破坏性关节病, 多继发于脊髓空洞症, 常导致肩关节骨溶解与严重的上肢功能障碍。其诊疗难点不在于单纯识别关节破坏, 而在于早期症状缺乏特异性, 易与感染、肿瘤及炎症性关节病混淆; 同时, 肩胛盂与肱骨头骨破坏程度差异较大, 传统的影像学描述难以为术前评估和治疗决策提供直接依据。NGH(neuropathic gleno-humeral degeneration)分类基于X线平片分别独立评估肩胛盂与肱骨头的退变程度, 有助于减少影像描述的模糊性, 为临床手术决策提供参考。现有证据提示, 夏科氏肩关节病的临床处理应建立在综合评估的基础上: NGH分类辅助X线可用于初步诊断, 磁共振成像(magnetic resonance imaging, MRI)可评价软组织等, CT+3D有助于骨缺损量化和假体规划; 存在肿胀、积液、炎症指标升高或拟手术者, 应结合关节穿刺、活检排除感染、肿瘤和炎症性疾病。对于关节破坏进入静止期的患者, 可采取肩关节置换术等手术。未来仍需开展多中心、前瞻性队列研究及长期随访, 以进一步验证NGH分类的临床价值, 并明确肩关节置换术的长期疗效。

[关键词] 夏科氏肩关节病; NGH分类; 脊髓空洞症; 影像学评估; 肩关节置换术

Imaging assessment and treatment of Charcot arthropathy of the shoulder assisted by NGH classification

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ABSTRACT

Charcot arthropathy of the shoulder is a rare destructive joint disorder that is most commonly secondary to syringomyelia and often leads to osteolysis of the shoulder joint and severe upper limb dysfunction. The diagnostic and therapeutic challenges of this condition lie not merely in identifying joint destruction, but in the nonspecific nature of its early clinical manifestations, which may mimic infection, tumor, or inflammatory arthropathy. In addition, the extent of bone destruction in the glenoid and humeral head may vary considerably, making conventional imaging descriptions insufficient for preoperative assessment and treatment decision-making. The neuropathic gleno-humeral degeneration (NGH) classification independently evaluates the degenerative changes of the

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glenoid and humeral head on imaging. This classification helps reduce ambiguity in radiographic descriptions and provides a reference for clinical surgical decision-making. Current evidence suggests that the management of Charcot arthropathy of the shoulder should be based on comprehensive assessment: Radiography may be used for initial diagnosis with assistance from the NGH classification; magnetic resonance imaging can evaluate soft tissue conditions and related abnormalities; and computed tomography with three-dimensional reconstruction is useful for quantifying bone defects and planning prosthetic implantation. In patients with swelling, joint effusion, elevated inflammatory markers, or planned surgery, joint aspiration and biopsy should be considered to exclude infection, tumor, and inflammatory joint diseases. For patients whose joint destruction has entered a quiescent phase, shoulder arthroplasty and other surgical procedures may be considered. In the future, multi-center and prospective cohort studies, and long-term follow-up are still needed to further validate the clinical value of NGH classification and clarify the long-term efficacy of shoulder replacement surgery.

KEY WORDS Charcot arthropathy of the shoulder; NGH classification; syringomyelia; imaging assessment; shoulder arthroplasty

夏科氏肩关节病是一种因神经感觉缺陷而发生的慢性关节疾病^[1-2]。其主要病因为脊髓空洞症(syringomyelia, SM),其他病因包括梅毒、糖尿病及创伤等^[3]。无痛性肿胀、关节活动度受限、肌力减弱等症状是该病的临床早期表现,中晚期可出现部分或全部感觉丧失、肩关节脱位及骨结构破坏等。

夏科氏肩关节病的临床处理目前面临多重困难。第一,早期症状不典型,患者容易被误诊为感染、肿瘤或炎症性关节病^[1,4-5]。第二,多数患者发现时已存在肱骨头和肩胛盂不同程度骨破坏,单纯使用“骨溶解严重”或“关节破坏明显”等描述难以指导后续影像评估和术前讨论。第三,由于夏科氏肩关节病发病率极低,且手术治疗证据主要来自病例报告,术式选择不能简单套用普通肩关节退变性疾病的治疗逻辑。

基于上述问题,本文介绍夏科氏肩关节病的主要发病机制、诊断路径及手术策略,重点聚焦 NGH(neuropathic gleno-humeral degeneration)分类系统(该分类可根据影像学结果对不同损伤程度患者进行分层,独立评估肩胛盂与肱骨头的退变程度)辅助下的夏科氏肩关节病影像学评估与治疗,以期临床诊断与综合治疗策略提供参考。

1 夏科氏肩关节病的流行病学特征与发病机制

1.1 流行病学特征

一项纳入 39 篇病例报告(共包含 65 例患者, 71

肩)的系统评价^[6]显示,患者的发病年龄为(51.1±15.8)岁,女性略多于男性(女:男=1.2:1)。受累关节以单侧为主(90.8%),左右两侧无显著差异,且绝大多数患者伴有关节活动度(range of motion, ROM)受限。27%的患者有创伤史,与无创伤史患者相比,诊断时间缩短 69%^[1]。

1.2 发病机制

夏科氏肩关节病是一种神经性关节病,多继发于 SM。SM 可继发于 Arnold-Chiari 等先天畸形,或由恶性肿瘤、脊髓损伤等因素引起^[7]。Chiari I 畸形、SM 和夏科氏肩关节病三者间的关系是临床上最主要的发病机制。

从分子机制来看,在夏科氏足(另一种神经性关节病)中,骨保护素(osteoprotegerin, OPG)、NF-κB 受体活化因子(receptor activator of NF-κB, RANK)及 NF-κB 受体活化因子配体(receptor activator of NF-κB ligand, RANKL)三者所构成的通路会引起严重的骨吸收。骨吸收同样为夏科氏肩关节病的特征之一,故 OPG/RANKL/RANK 信号通路可能作为外推机制,为夏科氏肩关节病中可能存在的分子发病机制的研究提供参考。

1.2.1 Chiari I 畸形继发 SM 致夏科氏肩关节病的发病机制

Chiari I 畸形(患病率为 3/10 万~8/10 万)主要特征为小脑扁桃体下疝,患者常伴头痛、肢体麻木及四

肢轻瘫等症状^[8-10]。小脑扁桃体下疝阻塞第四脑室出口,导致脑脊液循环受阻,异常压力传导至脊髓中央管,使65%~85%的患者在颈段或颈胸段(最常为C₅~T₁节段)形成纵行液腔,即SM^[11-13]。空洞扩张破坏了脊髓前角运动神经元及前角间束内的交叉温痛觉纤维,导致患者出现上肢肌力减退及典型的分离

性感觉障碍。20%~30%的SM患者会发展为夏科氏肩关节病^[4, 6, 11, 14]。其主要机制为:肩关节失去感觉后,患者无法及时察觉日常活动中的反复创伤,如脱位、半脱位、骨折等^[4]。由此进入失去神经保护的恶性循环,炎症激活、骨吸收增强、碎片形成、异常重建,最终导致关节破坏、肿胀、畸形(图1)。

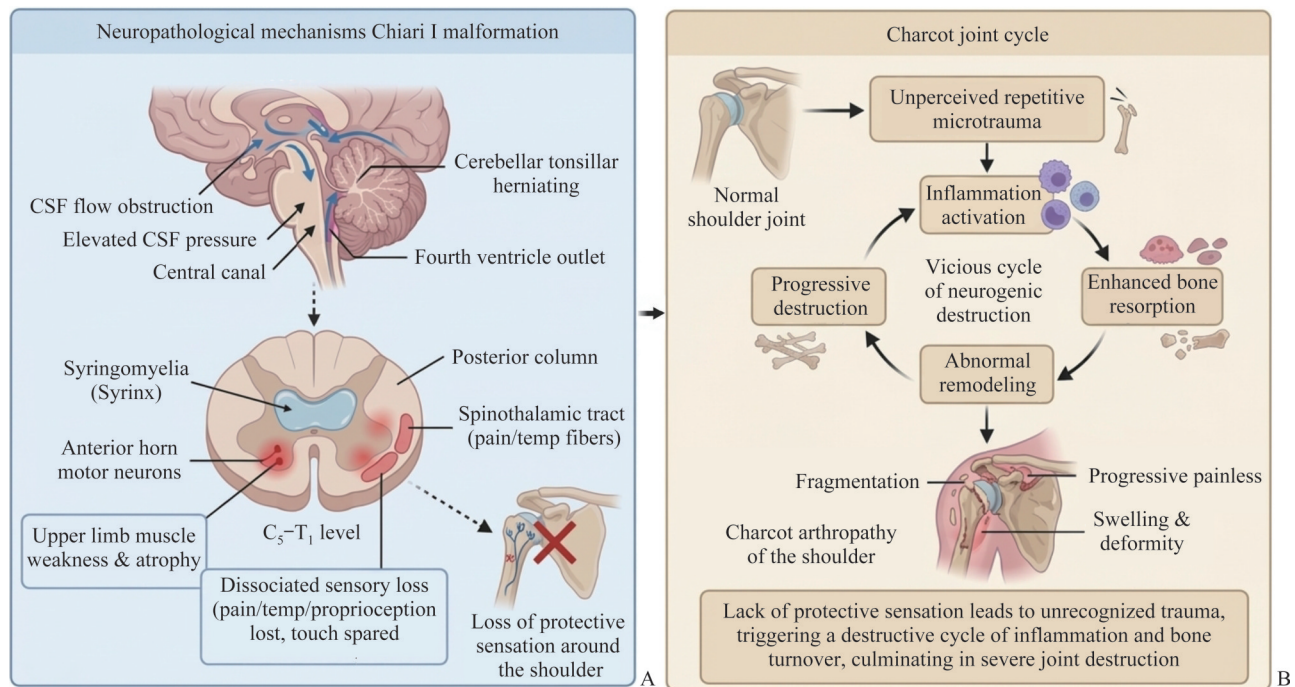


图1 Chiari I畸形继发脊髓空洞症在夏科氏肩关节病中的发病机制

Figure 1 Pathogenesis of syringomyelia secondary to Chiari I malformation in Charcot arthropathy of the shoulder

A: Chiari I malformation may obstruct cerebrospinal fluid flow and cause cervical syringomyelia, resulting in dissociated sensory loss, upper-limb weakness, and loss of protective sensation around the shoulder. B: Unrecognized repetitive microtrauma initiates inflammation, bone resorption, and abnormal remodeling, creating a destructive cycle that ultimately leads to painless swelling, deformity, fragmentation, and severe shoulder joint destruction. CSF: Cerebrospinal fluid.

1.2.2 骨吸收一般分子机制

在维持局部骨稳态的生理过程中,OPG/RANKL/RANK信号通路在调控骨重建中发挥关键作用^[2, 15]。局部病变可引发反复的微小创伤,激活OPG/RANKL/RANK信号通路,使骨质被过度吸收,最终导致局部骨质破坏(图2)。

正常生理情况下,破骨细胞和成骨细胞在骨折断端参与骨质修复,完成骨重建。成骨细胞分泌的OPG可竞争性结合RANKL,抑制破骨细胞活性并促进其凋亡,平衡骨密度及强度^[2, 16]。在病理状态下该通路失衡,具体表现为RANKL过表达,破骨细胞引

起局部骨质的持续破坏。RANKL与RANK相互作用,促进破骨细胞成熟与分化,加重炎症反应,引起骨吸收和骨量减少,进而导致骨质疏松与骨折。

一项2017年的临床观察性研究^[17]显示,夏科氏足的特异性标志是RANKL/OPG比值升高。然而目前暂无夏科氏肩关节病与OPG/RANKL/RANK信号通路的直接机制研究,仅有文献^[18]提到RANKL可能参与夏科氏肩关节病的发生。因此,OPG/RANKL/RANK通路可作为解释骨吸收的可能机制之一,尚不能作为已被证实的肩关节特异性发病机制。

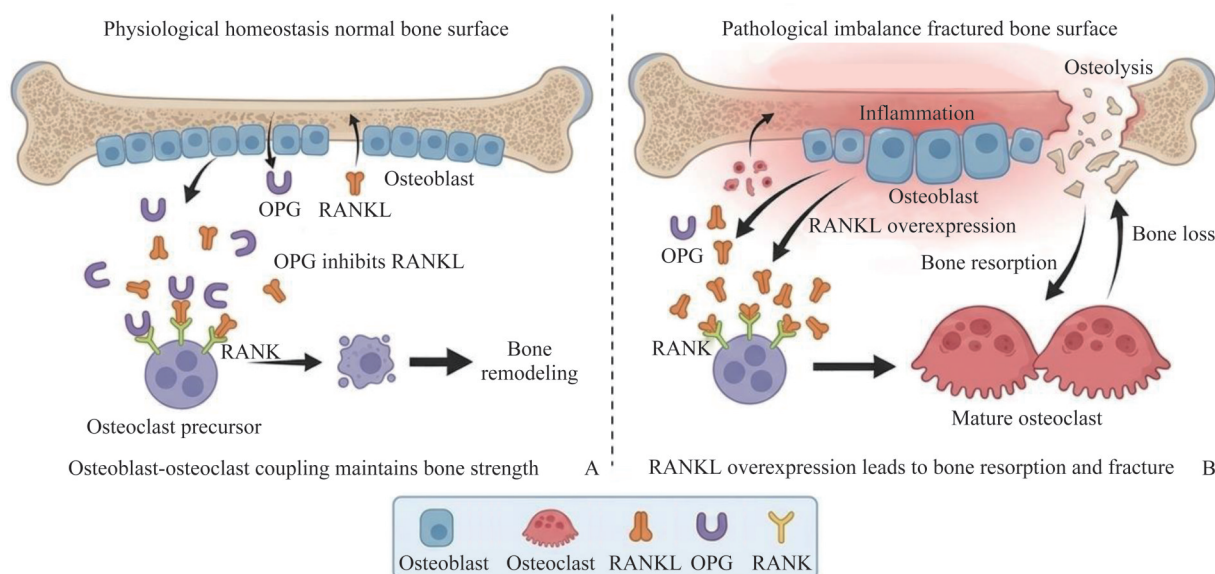


图2 OPG/RANKL/RANK信号通路调控骨吸收的作用机制示意图

Figure 2 Schematic diagram of the pathogenesis of OPG/RANKL/RANK signaling pathway in bone resorption

A: Under normal physiological conditions, osteoblasts and osteoclasts achieve balance and complete bone remodeling. B: Under pathological conditions, RANKL is overexpressed, and osteoclasts act too strongly, resulting in bone resorption. OPG: Osteoprotegerin; RANKL: Receptor activator of NF- κ B ligand; RANK: Receptor activator of NF- κ B.

2 夏科氏肩关节病的诊断

2.1 影像学评估

2.1.1 X线诊断与NGH分类

X线检查是评估肩关节骨性结构最常用的影像学手段,可直观显示关节间隙、骨质硬化及骨溶解等退变征象。依据多项回顾性研究^[4, 14, 19],夏科氏肩关节病经典影像学特征为“6D”征象:关节严重破坏(destruction, 肱骨头及肩胛盂骨质溶解)、脱位或半脱位(dislocation/subluxation)、关节内大量骨碎片(debris)、骨密度增加伴硬化(density increase)、关节紊乱(disorganization, 间隙不规则或消失)、软组织肿胀伴积液(distention),少数患者还可见新骨形成、积液及异位骨化等表现。早期仅有轻度软组织肿胀及骨质硬化,晚期破坏明显,且临床上常有无痛性肿胀这一矛盾表现^[20-21]。在一项小样本研究^[14]中,受累肩关节普遍表现为肱骨头完全或近乎完全溶解,而肩胛盂退变程度差异较大。这一发现说明夏科氏肩关节病的影像学特征不应仅概括为“严重关节破坏”,而应分别描述肱骨头与肩胛盂退变情况,为后续CT评估、假体固定可行性判断等提供依据。

Wawrzyniak等^[22]于2022年首次提出新的影像学分类,即NGH分类,该分类可独立评估肩胛盂(glenoid, G)和肱骨头(humeral head/proximal humerus, H)的退变程度,将二者各分为3个阶段:

G0/H0(无明显改变)、G1/H1(部分退变)和G2/H2(完全退变)(图3)。在该分类中,H2阶段最常见(约75%),而G阶段有一定变异。该分类基于2个投照位(以前后位为主)的X线平片,定性地评价骨质溶解程度及结构完整性。Wawrzyniak等^[6]于2023年发表的相关系统评价中进一步补充了病例资料,但目前仍缺乏前瞻性验证及与术后功能、假体生存率和并发症之间的结局关联研究。笔者认为,NGH分类的主要价值在于减少影像学描述的模糊性,并促进骨科、影像科和神经外科之间的术前沟通,但目前尚不能将其作为直接决定术式的确定性依据。

2.1.2 肩部磁共振成像

磁共振成像(magnetic resonance imaging, MRI)可进一步支持夏科氏肩关节病的诊断。MRI能清晰地显示关节退变程度、软组织是否改变及并发症,并有助于排除感染、肿瘤及退行性关节炎等其他疾病,避免误诊^[23]。夏科氏肩关节病典型的MRI表现有:肱骨头及肩胛盂骨质吸收,肱骨头可完全或近完全消失,呈截肢后样外观,肩胛盂亦不规则变形^[18, 24-25]。另外,常伴大量关节积液、关节囊扩张及周围软组织肿胀^[4, 11, 18]。广泛滑膜增生可形成巨大软组织肿块,内见关节内游离体或骨碎片^[18, 26]。通过MRI检查可进一步确诊,并确认肩胛盂和肱骨头的退变程度,为后续治疗提供参考依据。

MRI还可显示夏科氏肩关节病患者可能存在的

神经性原发病。一项2019年的系统回顾^[1]中, 76%的患者经MRI确诊有SM, 累及 (7.5 ± 4.5) 个节段。因此, 对于存在无痛性肩部肿胀、肌力下降、肌萎缩等神

经系统表现的患者, 笔者建议在肩关节MRI检查基础上, 加行头颅及颈胸段脊髓MRI, 以明确是否存在SM等神经性病变^[27]。

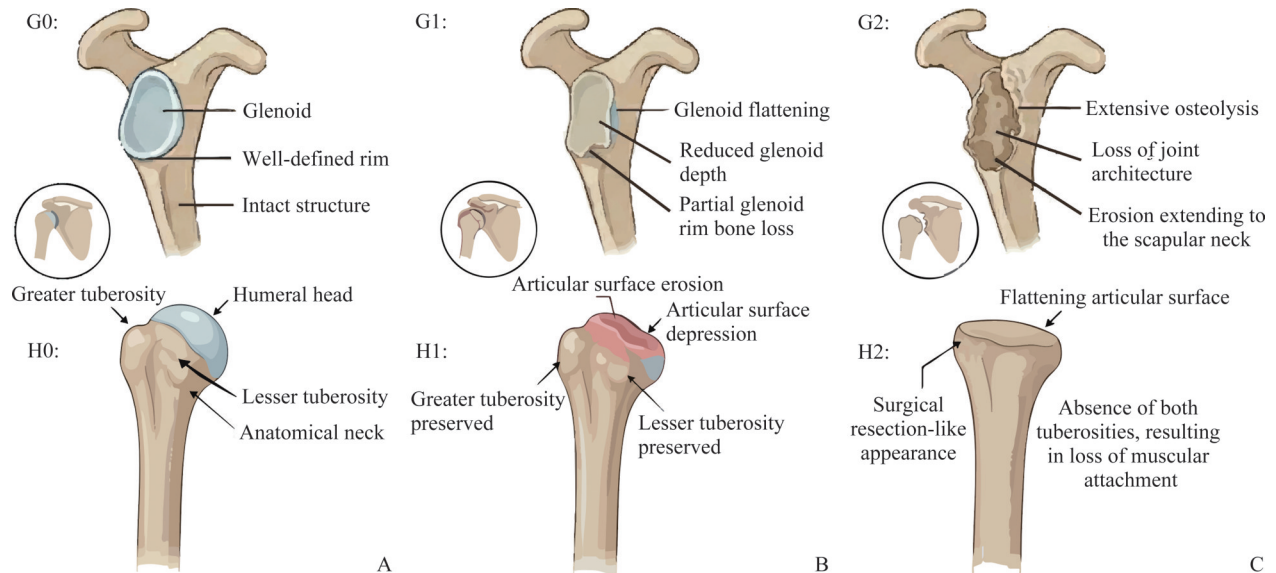


图3 NGH分类下评估肩胛盂与肱骨头的退变程度

Figure 3 Evaluation of the degenerative degree of the glenoid and humeral head under the NGH classification system

A: G0/H0. G0: Normal glenoid morphology and bone density; H0: Shape of the humeral head is intact, and the bone mass is normal. B: G1/H1. G1: Partial degeneration of the glenoid, partial bone erosion, sclerosis or mild absorption; H1: Humeral head is partially degenerated/absorbed, but the greater and lesser tuberosities still retain. C: G2/H2. G2: Glenoid is completely degenerated, and the bone is completely or nearly completely dissolved/disappeared; H2: Humeral head is completely or nearly completely degenerated and absorbed, and the tuberosity disappears or cannot be identified. NGH: Neuropathic gleno-humeral degeneration.

2.2 其他诊断方法

夏科氏肩关节病在急性破坏期可伴有显著的肩部红肿与关节积液, 极易被误诊为感染、关节炎等。笔者推荐采取如下排除流程: 1) 无菌穿刺与滑膜液分析。对患者进行无菌关节穿刺, 可进行细胞计数与分类、细菌培养及病原学筛查等, 以排除急性关节炎或结核分枝杆菌等细菌感染^[28-29]。2) 滑膜/骨组织活检。若难以排除恶性肿瘤等, 需进行滑膜及骨破坏区穿刺活检。若仅存在碎片性滑膜或骨质坏死而无恶性增殖细胞, 方可排除肿瘤等^[30-31]。

CT可在骨缺损评估及术前假体规划中发挥重要作用。患者重建前常进行患侧肩关节薄层CT平扫及三维(three-dimension, 3D)重建, 不推荐仅凭二维(two-dimension, 2D)平片进行术前规划^[32-33]。3D-CT能够排除关节脱位对骨性边界的遮挡, 既可量化肩胛盂骨缺损范围及深度, 又可测量肩胛盂后倾角, 为假体置入规划提供参考^[34-35]。

3 夏科氏肩关节病的治疗

3.1 保守治疗与对原发病的治疗

夏科氏肩关节病确诊时多已处于晚期, 手术治疗可能效果不佳, 通常以保守治疗为主, 即先控制炎症、缓解疼痛, 再进行物理治疗以适当改善功能^[36-37]。主要保守治疗包括限制负重、佩戴功能性支具、口服非甾体抗炎药减轻滑膜炎症等措施^[11]。

部分药物可治疗夏科氏足及部分以骨吸收为特征的关节疾病。地舒单抗是一种抗RANKL单克隆抗体, 可抑制RANKL对破骨细胞的刺激作用, 从而抑制过度的骨吸收^[38]。阿仑膦酸钠、帕米膦酸钠等双膦酸盐对夏科氏足的治疗有一定研究基础, 其机制可能为双膦酸盐通过抑制过度活跃的破骨细胞, 延缓夏科氏足的急性期进展^[3, 39]。双膦酸盐及地舒单抗理论上具有抑制骨吸收的作用, 但由于缺乏肩关节特异性的随机对照试验, 故目前不推荐作为夏科氏肩

关节病的常规治疗药物^[4]。被动牵伸、活动度训练及肌力强化等方法亦有利于减轻疼痛、减少肿胀^[20]。保守治疗可缓解患者疼痛,但不能从根本上解决患者ROM受限的问题^[1, 6, 14]。

局部骨科手术后可能出现神经支配永久缺失、软组织条件恶化,故单纯进行局部骨科手术极易出现假体松动、脱位、感染等并发症,手术失败率极高。因此,早期对SM进行神经外科干预(如针对Chiari I畸形的后颅窝减压术等)可能稳定甚至改善关节病变^[40]。已有个案及有限随访数据^[11]提示,在成功实施脑脊液减压引流后,部分患者的肩关节破坏进程得以实质性停滞。Makihara等^[41]报道的单个病例显示,在针对SM行枕下减压术后,晚期夏科氏肩关节病患者的严重肩胛盂骨缺损出现一定程度的骨质再生。由于该证据仅来源于个案报道,尚不能据此推断神经外科干预可稳定或逆转患者的肩关节破坏进程。但该病例提示,明确并处理原发神经病变可能是控制局部关节病变进展的重要前提。因此,对于疑似存在神经性病变的患者,建议在制订肩关节局部治疗方案前,优先完成神经系统评估,并与神经外科共同判断原发病是否需要干预^[31, 42]。

若为罕见的糖尿病继发的夏科氏肩关节病,治疗上首先要严格控制原发病,即在内分泌科指导下控制糖化血红蛋白(hemoglobin A1c, HbA1c),由此改善高血糖所致的微血管缺血、周围神经脱髓鞘病变,降低进一步神经功能损害的风险^[43-44]。

3.2 NGH分类辅助下的夏科氏肩关节病手术治疗

当患者经保守治疗无效,疼痛持续或功能障碍严重,且影像学与临床表现均显示关节病变已进入稳定期时,可考虑手术治疗。目前夏科氏肩关节病的手术治疗主要包括肩关节置换术、肩关节融合术以及关节镜冲洗和清创术,部分患者接受了联合手术治疗^[1, 45]。

NGH分类将患者进行分类与管理,为手术提供了一定参考。但实际在临床中,术式选择并非与NGH分类一一对应^[46]。术式选择应考虑多方因素:首先,患者已排除了感染等风险,且原发病已控制稳定^[47];其次,需判断肩胛盂和肱骨头的骨量能否实现可靠固定,并评估肩袖和三角肌功能是否良好^[48-49];另外,还需考虑患者依从性及术后康复能力等。综合评估后可谨慎进行手术(图4)。

3.2.1 全肩关节置换术

全肩关节置换术(anatomic total shoulder arthroplasty, TSA)中用金属球头配以聚乙烯盂球同时置换肱骨头

及肩胛盂以重建原生关节结构,可充分缓解疼痛、恢复功能。基于2025年的叙述性综述^[50],手术时机的选择需满足以下条件:疾病处于稳定期;影像学已证实关节全面退变,但肩袖无明显撕裂;患者年龄适中(通常>50岁),且能主动、充分地配合术后康复。在此基础上,宜在症状显著影响日常生活前尽早干预,以尽可能延长假体使用时间。处于NGH分类中G0/G1、H1阶段的患者,其影像学表现虽提示具备一定手术基础,但上述分级结果不能单独作为TSA适应证^[6, 22]。TSA的应用需十分严格,只有当肩袖肌群完整、肩胛盂骨量充足时才可选择,否则失败风险极大^[51]。

3.2.2 反式肩关节置换术

该手术通过反转肩关节球窝结构,将球状假体固定于肩胛骨侧,依赖三角肌提供外展力,增强肩关节机械稳定性,缓解夏科氏肩关节病患者肩关节功能受限问题。NGH分类中的G0/G1、H2阶段的患者可提示其具备一定手术基础^[6, 14, 22, 45]。但仅在原发神经病变相对稳定、三角肌功能良好、关节具备一定稳定性且肩胛盂骨量允许基座可靠固定的严格筛选病例中,将反式肩关节置换术(reverse shoulder arthroplasty, RSA)作为可选方案之一,可在一定程度上增强肩关节稳定性,并在部分患者中缓解疼痛、改善功能^[50]。

3.2.3 半肩关节置换术

该手术仅置换病变的肱骨头,用金属假体替代肱骨头的球状部分,同时保留或适度修整肩胛盂以恢复关节稳定性,但术后可能存在疼痛残留、关节不稳及功能改善有限的问题。根据2016年的系统评价^[45],手术建议时机为影像学上肱骨头退变严重而肩胛盂尚完整,通常于保守治疗失败且手术时机成熟后进行,以避免骨质溶解和畸形加重。当晚期患者肩胛盂骨质大量缺损(NGH分类提示患者处于G2阶段)、不能置入RSA基座固定螺钉时,半肩关节置换术(hemiarthroplasty, HA)是保留残余骨量、避免假体侧松动风险的折中方案^[6, 22, 52]。

3.2.4 肩关节融合术

历史上,肩关节融合术曾被用于重建畸形的夏科氏肩关节,但目前已很少应用。一方面,患者丧失了大部分痛温觉和本体感觉,即使实施牢固的内固定,患肢仍会在无意识下持续承受微小创伤;另一方面,局部失调引发关节异常充血,破骨细胞高度活跃,造成肩胛盂和肱骨头出现骨质溶解。在这种情况下,正常的骨愈合极难实现,手术常面临极

高的失败率, 并极有可能伴有并发症, 如骨不连、内固定物断裂及深部感染。因此, 肩关节融合术目

前已极少作为常规手段, 仅在置换术禁忌、其他治疗均失败的极少数晚期患者中, 可谨慎尝试^[12, 53]。

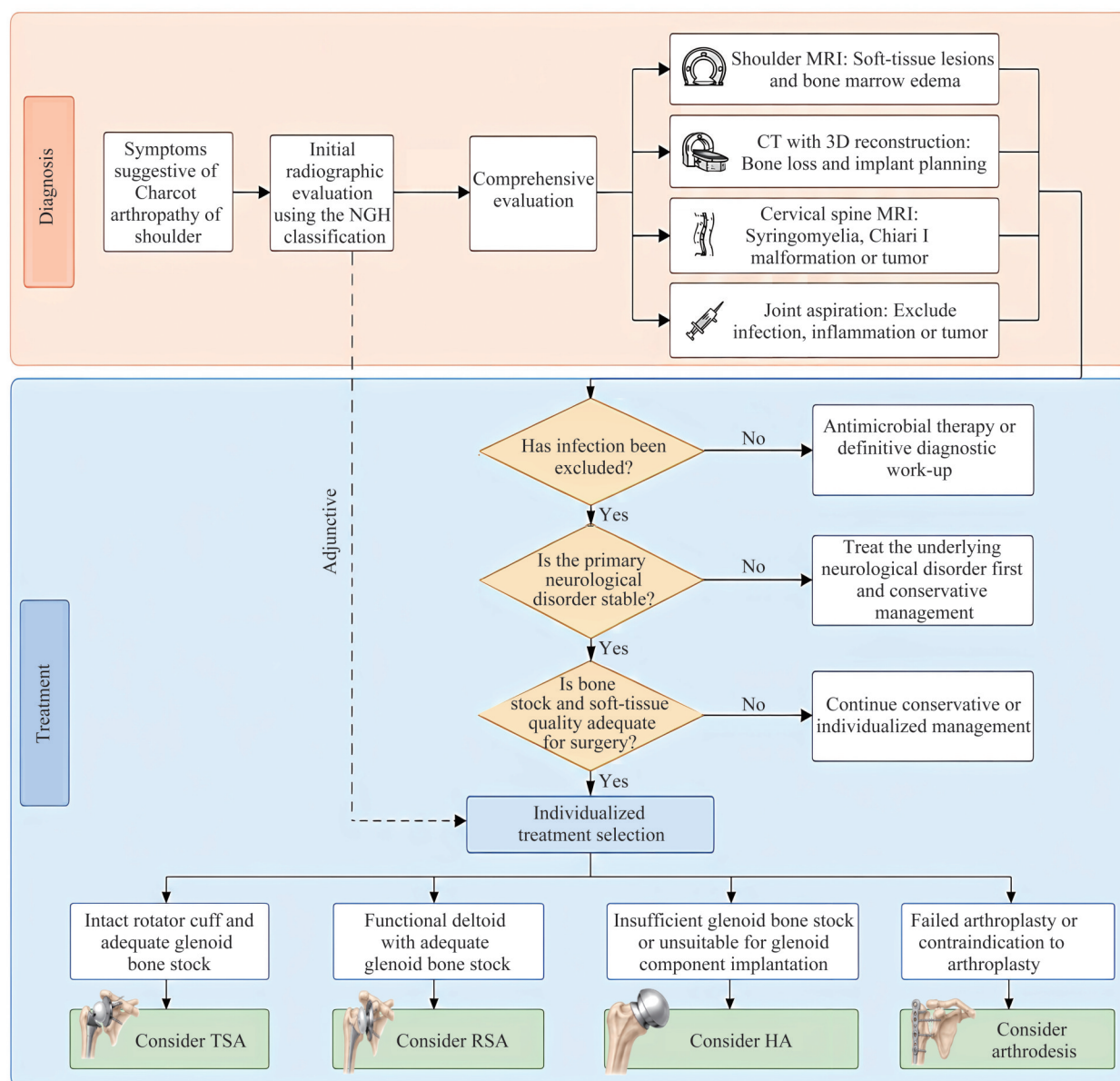


图4 夏科氏肩关节病诊疗决策建议流程图

Figure 4 Diagnosis and treatment decision-making flowchart for Charcot arthropathy of the shoulder

TSA: Anatomic total shoulder arthroplasty; RSA: Reverse shoulder arthroplasty; HA: Hemiarthroplasty; MRI: Magnetic resonance imaging.

4 结语与展望

NGH分类的引入, 从影像学角度为肩胛盂及肱骨头退变程度的评估提供了辅助工具, 为术式选择提供了一定参考。笔者认为, NGH分类的价值在于为夏科氏肩关节病提供更清晰的影像学分层语言,

但临床决策仍应建立在原发病是否稳定, 感染是否排除, 以及骨质条件和患者依从性的综合评估之上^[54]。

尽管对该病的病理机制与治疗策略的认识不断加深, 但现有的研究结论仍存在一定局限性。其一, 目前NGH分类尚不能单独作为术式选择的依据, 且

现阶段其证据主要来自病例报告和系统综述, 尚不足以单独预测术后功能、假体生存及并发症, 因此不能替代综合临床判断^[51]。其二, 在分子发病机制和药物保守治疗方面, 目前大量借鉴发病率更高的夏科氏足的研究结论, 但针对肩关节特异性的高质量证据依然缺乏, 其实际应用价值和临床疗效仍存在不足, 暂无法作为常规临床治疗指南推荐^[55]。其三, 虽然肩关节置换术短期内效果较好, 但由于样本量有限, 且夏科氏肩关节病患者存在保护性感觉缺失, 患者术后的假体生存率、无菌性松动率及并发症风险仍需长期随访加以验证^[24, 33]。

未来随着多中心、前瞻性队列研究及长期随访数据的不断积累, 有望进一步检验和完善NGH分类, 提高其准确性和适用性, 并持续优化夏科氏肩关节病的诊断与治疗策略。

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