

· 综述 ·

肱骨骨折术后骨不连巨大包壳形成：1例报告和综述[△]

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摘要: 骨不连是肱骨骨折术后较为常见的并发症, 指肱骨骨折后至少9个月内在3个月内没有骨愈合的迹象。肱骨骨折术后骨不连在老年患者中发生率较高, 导致患肢畸形和功能丧失, 严重影响患者日常生活与身心健康。既往文献多次报道了肱骨骨折术后骨不连的病因、危险因素、治疗手段和预后, 然而肱骨术后骨不连并巨大包壳形成在学术界却鲜有报道。本文报道本院创伤骨科收治的1例肱骨骨折术后骨不连巨大包壳形成, 分析其成因机制及治疗方案。此外, 本文对相关文献进行综述。

关键词: 肱骨骨折, 骨不连, 巨大包壳

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Nonunion complicated with huge cladding formation after plate fixation of humerus fracture: A case report and review // CHEN Shang-tong¹, CHEN Yue-ping², HUANG Chuan-hong¹. 1. Guangxi University of Traditional Chinese Medicine, Nanning, Guangxi 530000, China; 2. Department of Orthopedics and Hand Surgery, Ruikang Hospital, Guangxi University of Traditional Chinese Medicine, Nanning, Guangxi 530001, China

Abstract: Nonunion is a more common complication after humeral fracture, defined as no sign of bone healing for at least 9 months after humerus fracture. The incidence of nonunion after humeral fracture is high in elderly patients, resulting in deformity and loss of function of the affected limb, which seriously affects the daily life, as well as physical and mental health of patients. The etiology, risk factors, treatment and prognosis of postoperative nonunion of humerus fracture have been reported many times in the previous literature, however, postoperative nonunion of humerus complicated with a huge cladding formation has rarely been reported in the literature. In this paper, we report a case of nonunion complicated with a huge cladding formation after plate fixation of humerus fracture, and analyze its mechanism and treatment. In addition, this paper reviews the relevant literature.

Key words: humerus fracture, bone nonunion, huge cladding

肱骨骨折是临床一种常见骨折类型, 约占全身骨折的5%~10%^[1], 通常是指发生在肱骨外髁颈下1~2 cm至肱骨髁上2 cm的骨折^[2]。骨不连是肱骨骨折术后较为常见的并发症, 指肱骨骨折后至少9个月内在3个月内没有骨愈合的迹象^[3], 术后发生的概率为5%~10%^[4]。肱骨骨折术后骨不连在老年患者中发生率较高, 导致患肢畸形和功能丧失, 严重影响患者日常生活与身心健康。既往文献多次报道了肱骨骨折术后骨不连的病因、危险因素、治疗手段和预后, 然而肱骨术后骨不连并巨大包壳形成在学术界却鲜有报道。现报道本院创伤骨科收治的1例肱骨骨折术后骨不连巨大包壳形成, 并对既往的相关研究文献进行综述。

1 临床资料

患者女性, 80岁, 因“左上臂骨折术后9年, 畸形、疼痛活动受限5年”为主诉入院。体格检查: 患者左上臂中上段可见向前、向外突出畸形, 周围可见多处陈旧性手术瘢痕, 无明显渗出, 左上肢肌肉萎缩明显, 左上肢较右上肢短缩约2 cm。左上臂中上段处压痛明显, 压痛处可触及8 cm×4 cm肿物, 质地硬, 推之不移, 未触及骨擦感及异常活动。左肩关节外展上举90°, 后伸30°, 前屈上举90°, 内旋80°, 外旋40°, 水平前屈90°, 水平后伸30°。左桡动脉搏动良好, 左手各指血运、皮肤感觉及活动良好, 未见

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垂腕、爪形手、猿手等异常症状，余肢体查体未见明显异常。患者既往因外伤致左肱骨干骨折，术前X线片示骨折类型为12-C型（图1a），在当地医疗机构行左肱骨骨折钢板内固定术+钢丝捆扎术。第1次术后X线片检查提示：左侧肱骨上段骨折内固定术后内固定器松脱、骨髓腔内密度增高（图1b）；术后8年复查X线片均提示固定器松脱、骨折远端囊袋状异常密度影进行性增大、骨折断端无愈合倾向（图1c）。术后8年CT三维重建提示：左侧肱骨上段骨折内固定术后，骨折不愈合，金属内固定器下端松脱、左肱骨骨折远端前外侧囊袋状异常密度影（图1d）。患者多年来反复出现左上臂疼痛，左肩关节活动受限，近1年来上述症状再发加重，严重影响日常生活，于2022年8月以左肱骨骨不连收治于本院创伤骨科。结合病史、影像学及查体，符合左肱骨骨折术后骨不连并巨大包壳形成诊断，具备手术治疗指征。

排除禁忌证后行手术治疗。全身麻醉后，取仰卧位，消毒铺巾。沿左上臂原切口切开皮肤、皮下组织及深筋膜。显露钢板螺钉后见左肱骨内固定物松动，远端处钢板、钢钉连接松懈，部分螺钉脱出，使用改锥取出螺钉共10枚，取出钢板1块、钢丝2条。暴露骨折断端，检查骨折处可见断端对位差，骨折端硬化，近侧及远端骨质增生明显，远端形成类似假肱骨头的巨大包壳，内可见大量的大小约为0.3 cm×0.3 cm的白色圆球状颗粒。用咬骨钳清除断端之间的瘢痕肉芽组织，去除不规则状骨质、远端增生骨质，直至渗血为止，留取白色圆球状颗粒送病理检查；使用骨折复位钳使断端重新对位，开槽扩髓，置入髓内钉固定，远近端各拧入2枚螺钉，被动活动上臂见骨折断端无微动，透视下复位满意后冲洗术口，植入同种异体骨及骨修复材料后关闭切口，术毕（图1e, 1f）。术后1年复查X线提示骨折断端对线对位好，骨质密度增高、骨痂形成（图1g, 1h）。

术后病理提示：肉眼可见灰白囊壁样骨组织一块，大小7.5 cm×4 cm×3.5 cm，壁厚0.2~0.4 cm，内含大量米粒样物，直径0.2~0.5 cm。意见：符合骨折术后修复改变并多量游离体形成。镜检见少许破碎骨质、骨皮质不规则增厚；髓腔内显著硬化伴纤维化骨；另见部分游离粉染均质小体伴骨化、钙化。片内未见明确肿瘤证据。VG示髓腔纤维化。

2 讨论

2.1 发病机理与特点

目前国内外大量文献报道了肱骨骨不连的成因机制，但尚未有学者报道肱骨术后骨不连并巨大包壳形成。经查阅文献，本例巨大包壳的形成是基于术后骨不连所发生的，后者向前者转变，并逐渐形成较大的包壳样钙化颗粒沉积。正如病理所见，包壳镜检见破碎骨质、骨皮质不规则增厚，提示该包壳的形成机制与骨不连的机制类似，由骨不连发展而来。文献报道了肱骨骨折术后骨不连与机械不稳定、全身因素、血供破坏、骨祖细胞缺乏、骨痂重塑受损相关。骨折后机械不稳定导致断端反复运动，若局部组织应变超过一定水平，则导致纤维组织过度增生，阻碍骨形成。Stewart等^[5]认为，机械信号由细胞受体、离子通道和初级纤毛所感知，能激活钙信号通路、不同激酶通路以及Wnt/ β -catenin等信号通路，调控成骨分化。Lienau等^[6]发现，内固定失稳使得骨干进行剪切运动，导致骨折断端周围血供减少，6~9周后出现骨折断端的骨不连。Augat等^[7]认为，骨折断端微动能够促进骨折愈合，而水平方向的剪切运动会造成骨折处产生过度应变，导致骨膜中骨痂形成减少、骨折间隙中骨形成延迟。Claes等^[8]认为，应变<5%可促进骨折直接愈合、5%~15%的应变可促进软骨内>15%的应变诱导纤维组织形成；与压缩应变相比，剪切运动会造成骨折断端周围的血管化降低4倍，骨形成降低2倍^[9]；骨形成的降低、骨折间隙内纤维软骨组织增殖，造成骨不连的发生^[10]。Borgiani等^[11]发现，细胞机械信号的改变是导致老年小鼠骨不连的重要原因。杨国勇等^[12]认为，过早行上臂的旋转动作锻炼产生骨折端造成较大的剪切力、增加断端微动，亦会导致肱骨骨不连的发生。全身性因素主要与吸烟、饮酒、骨质疏松、糖尿病、肾功能不全、肿瘤、高血压等全身因素相关^[13]。长期吸烟释放的尼古丁及其他物质会影响正常骨组织的形成、代谢和血管再生，降低成骨细胞活性，降低骨硬度^[14]。过量饮酒会抑制新骨的形成与矿化，降低骨折断端稳定性，影响骨折愈合过程^[15]。骨质疏松的患者，成骨细胞与破骨细胞维持骨稳态的能力失衡，骨折修复能力较差，易导致骨不连的发生^[16]。高血压患者钙离子异常，影响骨折愈合^[17]。Choy^[18]、Clark等^[19]认为，全身的慢性炎症导致促炎细胞因子增加，加速细胞衰老，降低骨愈合能力，增加骨不连的风险。血供的破坏同样是骨不连发生的重要因素。Weiss等^[20]发现，骨不连的患者bFGF、PDGF-AB相较于正常骨折愈合的患者降低，二者参与调控血管的生成与骨折愈合。高能损伤作用于肱骨，导致软组织损伤、皮肤大范围缺

损、滋养动脉破坏，以及骨片游离，破坏了肱骨断端周围的生物学环境，导致肱骨骨不连的发生^[21]。Cha 等^[22]认为，相较于髓内钉，钢板治疗肱骨干骨折，骨不连的发生概率更高，原因在于钢板固定轴向稳定性差、术中骨膜广泛剥离、损伤滋养动脉，影响骨折愈合。Orapiriyakul 等^[23]提出，选用髓内钉治疗肱骨骨折，扩髓时会导致髓腔内血供破坏、摩擦产热加重缺血性骨坏死发生的概率。Duchamp 等^[24]认为，骨祖细胞能够促进软骨和骨的生成，影响骨折愈合与塑形，骨祖细胞缺乏也是骨不连的原因之一^[25]。骨祖细胞来源于骨膜和肌肉等软组织，因此严重软组织损

伤导致骨不连与骨祖细胞的丢失密切相关^[26]。骨痂重塑受损是骨不连又一重要因素。骨折断端接触能够影响骨痂的形成，由于缺乏硬骨痂的桥接会导致无法进入骨折的塑形期。孙月华等^[27]认为，骨折接触面积越大，所承受的应力越小，受到的应力干扰小，越倾向于骨折愈合。宣勇等^[28]认为，由于肱骨远端变平，置入髓内钉会牵张骨折断端，增大断端间隙，增加骨不连的风险。粉碎性骨折、多段骨折以及骨缺损减少造成骨折断端接触不良，成骨细胞难以通过骨折间隙，无法形成有效骨痂，内固定物的应力持续增加，最终增加骨不连的风险^[29]。

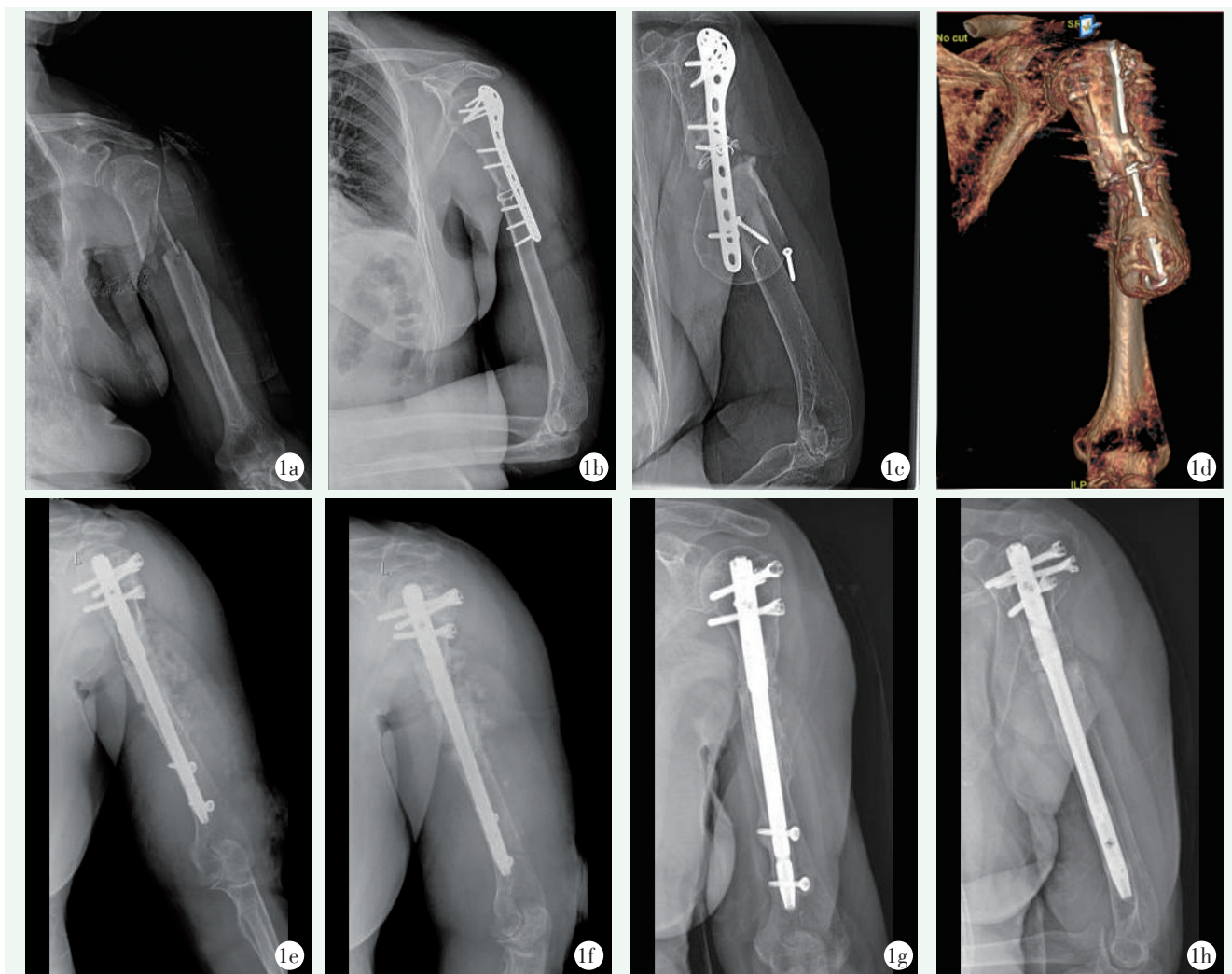


图 1. 患者女性，80 岁。1a: 术前 X 线片示骨折类型为 12-C 型；1b: 术后 X 线片显示左肱骨骨折钢板内固定术+钢丝捆扎术；1c, 1d: 术后 8 年 X 线片和 CT 显示钢板松动、巨大包壳影，骨折不连；1e, 1f: 行肱骨交锁髓内钉内固定+植骨术翻修术后，X 线片示内固定位置良好；1g, 1h: 翻修术后 1 年，X 线片提示骨折断端对线对位好，骨质密度增高、骨痂形成。

Figure 1. A 80-year-old female. 1a: Preoperative radiograph showed type 12-C fracture; 1b: Postoperative X ray revealed the fracture fixed with plate screws and cerclage wire; 1c, 1d: Radiograph and CT 8 years after surgery showed the implants breaking and loosening, complicated with a huge cladding shadow, and nonunion of the fracture; 1e, 1f: After the revision surgery of humeral interlocking intramedullary nail fixation and bone grafting, X-ray films showed the implants of internal fixation in good position; 1g, 1h: Radiographs a year after revision showed good humeral alignment with callus formation.

本例病例合并巨大包壳形成，其原因可能在于术后负荷的增加。术后负荷的增加，导致螺钉进一步松动，在三角肌收缩下引起骨折远端向外上方移位，自下而上撬拨钢板，增加了钢板与骨面的间隙，摩擦力减小，骨折断端对线对位差，增加骨不连的发生概率。由于远端螺钉的松动，失稳的钢板以近端螺钉为支点在体内产生“钟摆样”运动，运动产生的机械刺激能够诱导软骨发生^[30]、细胞分裂和炎症相关的基因表达^[31]，促进成骨分化^[32]。新生的骨组织会通过增加或减少局部骨孔隙度，调整局部骨小梁方向，向应力刺激的方向进行骨生长重塑^[33]，最终达到稳定状态，形成巨大包壳。该结果与 Kristy、Marcucio 等^[34, 35]的观点类似。本例病例的术后病理结果提示，囊袋状物为破碎骨质，而非肿瘤细胞的异常增生，同样证明了上述观点。

2.2 临床表现

肱骨骨折术后骨不连巨大包壳的临床表现与骨不连类似。肱骨骨不连最主要的临床表现为上臂畸形、骨折断端异常活动，伴有局部疼痛、压痛及叩击痛，疼痛常常随着活动和负重的增加而加重^[36]。随着病程时间的延长，部分骨不连的患者疼痛会逐渐减轻，直至消失。张宏峰等^[37]认为，肱骨骨折术后骨不连最典型的临床表现是功能丧失，Jupiter 等^[38]认为，肱骨骨不连的功能障碍以肩关节和肘关节僵硬为主。Nicholson 等^[39]认为，骨不连患者不仅患肢受限，且通常合并严重的心理疾病。影像学表现主要为骨折线清晰但无连续骨痂通过，骨折断端分离、硬化、间隙增大，髓腔封闭。X 线下肥大型骨不连通常可见骨反应明显、骨质硬化，类似大象腿或马蹄形大而宽的骨痂形成，但断端有间隙；萎缩性骨不连的骨折间隙周围有少量骨痂形成，断端吸收变细，周围缺乏骨膜反应，缺少血供^[40]；而营养不良型骨不连 X 线片示断端周围几乎无骨痂形成或少量骨痂形成^[41]。假关节性骨不连 X 线片示骨折断端髓腔封闭被纤维软骨覆盖，并且形成假关节^[42]，Gregory 等^[43]认为，8%~12%的肱骨干骨折患者合并假关节的形成。

2.3 治疗方法

肱骨骨折术后骨不连的治疗原则是为断端提供足够的机械稳定以及恢复断端生物学支持。肱骨骨折术后骨不连通过手术治疗可以取得较好的临床效果。主流观念认为，无菌性肱骨骨不连手术治疗包括骨移植和内固定。对于稳定性高、固定牢靠、骨丢失少的骨不连类型，骨移植能够提高术后治愈率。骨移植的金标准是自体骨移植，具有成骨能力强、生物学活性

高、免疫排斥风险低等优势，但受限于供区，通常情况下难以满足手术所需的骨量^[44]。异体骨、人工骨移植能够解决骨量不足，但其生物学活性差，在骨愈合过程中仅起传导作用，并且使用异体骨移植仍存在医学伦理争议。吴敏等^[45]采用皮质剥脱术联合皮质外骨桥技术治疗肱骨骨不连，能够有效诱导成骨、促进骨愈合。混合植骨^[46]、微创植骨^[47]、“J”形植骨^[48]等新植骨技术在临床中取得了较好的治疗效果。8 孔 4.5 mm 宽的加压钢板是治疗肱骨骨不连的金标准，其骨愈合率为 90%^[49]。应用锁定加压钢板可以最大限度的压缩骨折断端，增加拔出阻力^[50]。锁定钢板能够为骨不连处提供足够的机械稳定，把内固定的剪切力转换为螺钉与骨之间的压力，增强皮质骨对抗压缩负荷的能力。有研究表明，相较于髓内钉，钢板治疗肱骨骨不连痊愈率更高^[51]。Teciml 等^[52]认为，双钢板系统相较于单钢板结构更稳定，治疗肱骨骨不连在恢复肩关节和肘关节功能中更具优势，但该术式需要大量的软组织剥离，存在增加感染、再次骨不连和神经损伤的风险^[53]。髓内钉能够有效防止成角、旋转，提供旋转及垂直稳定。赵伟超等^[54]认为，采用髓内钉治疗肱骨骨不连扩髓不仅能够增加髓内钉与骨接触面积，并且碎屑能起到内植骨的作用，促进骨折的愈合。王志勇等^[55]认为，髓内钉联合钢板技术增加了断端旋转和成角稳定性，力学稳定性的增加有利于骨痂生成。李祖涛等^[56]认为，相较于钢板，Multiloc 髓内钉治疗肱骨干骨折骨不连，可以减少术中出血量、降低骨折愈合时间、提高肘关节功能。

在该病例中，采用肱骨交锁髓内钉内固定并植骨术进行翻修，原因在于：（1）本例患者骨不连的病程长，内外骨膜血供由于恢复时间久，相对稳定，有采用交锁髓内钉治疗的指征；（2）长时间的畸形愈合，很可能因肱骨结构改变，使得钢板无法紧贴骨面提供稳定固定，交锁髓内钉内固定术能够为骨折断端提供足够的稳定性，对于骨质疏松的患者，采用交锁髓内钉具有角度稳定的优势；（3）术中通过扩髓、凿腔，增大锁定螺钉与髓腔接触面积、提供的固定更为牢靠，能够有效防止术后再次出现内固定物失稳；（4）扩髓形成的骨屑位于骨不连部位的皮质骨内，与植入的异体骨能诱导成骨，修复骨折断端；（5）固定牢靠，使得患者能够尽早恢复功能锻炼，有利于肢体功能的恢复。术后患者骨折愈合情况、肩肘关节功能恢复均良好。

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