

骨质疏松性骨折围手术期干预指南

中华医学会骨质疏松和骨矿盐疾病分会骨与关节学组
中国医师协会骨科医师分会骨质疏松工作委员会

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骨质疏松性骨折系患者在日常生活中, 受到轻微外力或通常不会引起骨折的外力时发生的骨折, 亦称脆性骨折 (fragility fracture)。“通常不会引起骨折的外力”指在人体站立高度或低于站立高度跌倒时所产生的冲击^[1-2]。围手术期是指围绕手术的一个全过程, 从患者决定接受手术开始, 到手术治疗直至基本康复, 包括手术前、手术中及手术后的一段时间。具体是指从确定手术治疗时起, 直到与此手术相关的治疗基本结束为止, 时间约在术前 5~7 d 至术后 7~12 d。

骨质疏松性骨折不同于普通的创伤性骨折, 其发生与骨密度 (bone mineral density, BMD) 减少、骨微细结构破坏、骨质量下降等全身骨骼病理性改变密切相关^[3-5]。骨质疏松性骨折常见部位: 胸腰段椎体、髌部 (股骨近端)、腕部 (桡骨远端)、肩部 (肱骨近端)、骨盆和肋骨等^[6-9]。骨质疏松性骨折发生率随增龄上升, 65 岁以上人群发生率最高; 1992~2006 年中国北京地区, 85 岁以上人群髌部骨折男性增加了近 4 倍, 女性增加了近 5 倍^[10]。2013 年国际骨质疏松基金会

(International Osteoporosis Foundation, IOF) 报告: 全球每 3 秒钟就发生 1 例骨质疏松性骨折, 约 50% 女性和 20% 男性在 50 岁后会遭遇初次骨质疏松性骨折, 初次骨质疏松性骨折患者有 50% 将会发生再次骨质疏松性骨折^[1]。骨质疏松性骨折可引起疼痛和致残, 使患者生活质量显著下降; 髌部和椎体骨质疏松性骨折可降低患者预期寿命; 长期卧床的髌部骨质疏松性骨折高龄患者, 6~12 个月病死率高达 20%~30%, 永久性致残率超过 50%^[1,11-14]。

绝经后骨质疏松性骨折患者有 42%~56% 从未接受抗骨质疏松治疗, 当发生骨折后, 接受抗骨质疏松治疗的患者亦不足 20%; 另一方面, 骨科医师对骨质疏松性骨折重视程度严重不足, 欧洲多中心调查数据显示: 对初次骨质疏松性骨折患者进行骨密度检测的骨科医师不足 10%, 75% 的骨科医师对骨质疏松症基本概念认识不足^[12]。

为此, 中华医学会骨质疏松与骨矿盐疾病分会骨与关节学组和中国医师协会骨科医师分会骨质疏松工作委员会, 针对骨质疏松性骨折围手术期相关

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的临床评估、药物干预等常见临床问题（未涉及骨折具体手术治疗），组织相关专家，根据现有临床文献系统回顾分析，结合我国骨质疏松性骨折围手术期干预的现实情况，编写了本指南。

本指南目标人群：骨质疏松性骨折手术患者。本指南受众人群：参与骨质疏松性骨折临床诊疗工作的各级医护人员，包括骨科、急诊科、骨质疏松科、老年病科、内分泌科、放射科、康复科等；另外，保险人员、政府部门相关人员、卫生政策制定人员也可从本指南中获得相关信息。

本指南并非为临床疾病的诊断治疗标准，因为患者的实际情况存在个体差异；在临床实践中，骨质疏松性骨折患者病情存在独特性和差异性，未必完全与指南建议的情况一致，故医务人员在实际工作中可将本指南建议作为参考，根据患者个体情况进行独立判断和诊疗。

骨质疏松性骨折诊断

诊断骨质疏松性骨折应根据骨折临床表现、影像学检查、骨密度测定等结果，结合患者年龄、性别、既往骨折史及骨质疏松家族史等特点综合分析^[2,15-18]。

临床表现：骨质疏松性椎体骨折可有急性或慢性腰背痛、身高变矮、脊柱侧凸、驼背、胸廓畸形等临床表现，新发椎体骨折时通常伴有急性腰背部疼痛，慢性腰背部疼痛多见于陈旧性椎体骨折，部分患者还同时伴有胸闷、气短、呼吸困难等症状^[15-17]。骨质疏松性骨折发生在长骨中段时可见骨折特征性表现（畸形、骨擦感、反常活动）；骨折发生在长骨干骺端或脊柱椎体时，骨折特征性表现往往不典型^[15-17]。

影像学检查：典型骨质疏松性骨折 X 线影像学表现除了有骨折特征性影像特征外，还有骨皮质变薄、骨髓腔扩大、骨组织光透亮度增加、骨小梁稀疏等影像特征^[15-17,19-20]；CT 扫描或 CT 三维重建有助于判断骨质疏松性椎体骨折的粉碎程度和椎管破坏情况^[16,20-21]；磁共振（magnetic resonance, MR）检查有助于鉴别骨质疏松性骨折的“新鲜”与“陈旧”状态，且对

于隐匿性骨折、肿瘤转移性骨破坏有很好的鉴别诊断价值^[15,17,22-23]。

BMD 测定：BMD 是反映骨骼矿物质含量的一项客观指标，世界卫生组织（World Health Organization, WHO）和 IOF 推荐临床骨质疏松症诊断选用双能 X 线吸收检测法（dual energy X-ray absorptiometry, DXA），并强调 DXA 是临床骨质疏松诊断的金标准。临床可用于 BMD 检测的方法还有：外周双能 X 线吸收测量法（peripheral dual energy X-ray absorptiometry, pDXA），定量计算机断层照相术（quantitative computed tomography, QCT），外周定量计算机断层照相术（peripheral quantitative computed tomography, PQCT），定量超声法（quantitative ultrasound, QUS），双光子吸收测量法（dual photon absorptiometry, DPA），单光子吸收测量法（single photon absorptiometry, SPA）等^[8,15-17,20-21,24-27]。DXA 检测结果是骨质疏松症临床诊断和疗效评估的重要依据。骨折患者拟诊断骨质疏松性骨折时，若没有近期的 BMD 检测结果，建议进行 DXA 检测 BMD。骨质增生、陈旧性骨折、营养不良等患者自身因素可能会影响 DXA 检测结果，故在临床实际工作中，DXA 的检测结果还需结合患者实际状况，综合判断^[8,17,20-21,24]。

实验室检查：反映骨转换状况的骨代谢指标较多，IOF 和各国骨质疏松诊疗指南多推荐的骨转换指标（bone turnover markers, BTMs）包括骨形成指标，血清 I 型原胶原 N-端前肽（procollagen type I N-terminal peptide, PINP）；骨吸收指标，血清 I 型胶原 C-末端肽（serum C-terminal telopeptide of type I collagen, S-CTX）。骨转换指标可独立于 BMD 提示骨质疏松类型，预测骨折风险^[1,15-17,28-29]。

骨质疏松性骨折鉴别诊断

原发性骨质疏松性骨折与继发性骨质疏松性骨折鉴别诊断主要依靠实验室检查，包括血、尿常规，肝、肾功能，血钙、血磷、血碱性磷酸酶（alkaline phosphatase, ALP），25 羟维生素 D，性腺激素、甲状旁腺素（parathyroid

hormone, PTH), 尿钙、尿磷, 甲状腺功能, 皮质醇, 血尿酸等化验检查, 以及核素骨显像检查、肿瘤标志物 (tumor marker, TM) 检测等专项测定, 这些检测结果, 对原发性骨质疏松性骨折与继发性骨质疏松性骨折的鉴别判断有重要临床价值^[15-17, 20, 24]。

骨质疏松性骨折发生, 同时伴有 BMD 显著降低 (T 值或 Z 值均显著下降)、实验室检查结果明显异常, 应注重下列与骨骼相关的疾病鉴别: 肿瘤性疾病 (骨转移瘤、多发性骨髓瘤)、内分泌性疾病 (成骨不全、甲状旁腺功能亢进、库欣综合征、Paget 骨病、骨软化症)、免疫性疾病 (类风湿关节炎)、遗传性骨病、消化道和肾脏疾病 (影响钙与维生素 D 吸收调节功能) 等^[8, 15-16, 20, 24, 30-36]。

骨质疏松性骨折发生后, 除针对骨折的临床干预外, 还应详细了解患者既往疾病史、药物应用情况, 因为某些疾病或药物可诱发骨质疏松症导致骨折, 了解相关内容有助于骨质疏松性骨折的鉴别诊断^[16-18]。诊断为继发性骨质疏松性骨折后, 在骨折治疗的同时强调原发病的治疗和控制, 以及抗骨质疏松治疗^[16, 24, 37]。

骨质疏松性骨折危险因素

骨质疏松性骨折的主要危险因素^[16-17, 38-54]包括: (1) 过度瘦弱或肥胖; (2) 长期服用某些药物, 如糖皮质激素、抗凝剂、利尿剂、安眠药、抗惊厥药及影响神经系统类药物等; (3) 维生素 D 缺乏; (4) 既往有骨质疏松性骨折史; (5) 酗酒; (6) 长期吸烟; (7) 早期绝经; (8) 骨质疏松家族史等。

下列因素与骨质疏松性骨折存在相关性^[18, 24, 43, 55-60]: (1) 长期卧床或缺乏体育运动; (2) 频繁跌倒; (3) 某些先天性或遗传性骨病; (4) 吸收不良病史; (5) 患有 3 种以上慢性疾病; (6) 低钙摄入; (7) 绝经后铁蓄积; (8) 高盐摄入。

骨质疏松性骨折风险评估

风险评估是指应用 BMD 值、BTMs、简易评

估工具等方法, 预测骨质疏松性骨折发生的风险。

风险评估策略: 65 岁以上女性、75 岁以上男性应常规进行骨折风险评估^[3-5]; 患者年龄不足 50 岁存在下列因素时也应进行骨折风险评估: 应用糖皮质激素、闭经或早年绝经、频繁骨折史; 绝经后女性、65 岁以上男性股骨近端 BMD T 值低于 $-2.5 SD$ 者或骨转换指标异常者^[61-63]。

常用风险评估工具: 目前采用 FRAX[®] (<http://www.shef.ac.uk/FRAX/>) 预测骨质疏松性骨折风险的方法在国外已广泛应用, 但国人数据及其研究结果存在较多分歧, 可能低估了中国人骨质疏松骨折的风险^[15-16, 64-66]。

骨质疏松性骨折围手术期干预的临床思维

骨质疏松性骨折治疗原则: 复位、固定、功能锻炼、抗骨质疏松治疗^[15-16, 20, 67]。骨质疏松性骨折治疗目标可分为两个部分: 近期目标是改善临床症状、减少并发症, 远期目标是促进骨折愈合、功能康复、预防再骨折^[15-16, 20, 68-71]。

骨质疏松性骨折骨科治疗应强调个性化, 在综合评估患者全身状况、骨折部位、骨折类型、骨质疏松程度后选择手术或非手术治疗^[15-16, 20, 24, 67, 72]; 骨质疏松性骨折复位和固定应尽可能简单有效, 注重功能恢复; 采用内固定时应充分考虑骨质量差、骨愈合能力弱等特点, 仔细选择适宜的内植物、专有固定技术、内植物固定强化技术, 必要时应采用自体骨、异体骨、生物材料 (骨水泥、硫酸钙) 等物质充填骨缺损^[15-16, 20, 24, 70, 72-74]。

骨质疏松性骨折采用外固定术治疗时, 应评估骨折部位软组织状况, 采用轻便有效、对功能康复影响小的外固定方法^[15-16, 24, 67, 72, 75]。

骨质疏松性骨折患者有开放手术适应证时, 应尽快评估患者全身及骨骼状况, 尽可能早期实施手术; 对高龄骨质疏松性腕部骨折患者, 不推荐术前骨牵引; 正在应用抗血小板药物 (阿司匹林或氯吡格雷) 治疗的患者不推荐延迟手术; 全身麻醉或椎管内麻醉对预后影响无差异; 应用预防静脉血栓治疗可有效降低深静脉血栓 (deep ve-

nous thrombosis, DVT)、肺栓塞 (pulmonary embolism, PE) 的发生; 对营养状况较差患者尽早给予营养支持以降低术后病死率^[67,76-77]。

骨质疏松性骨折围手术期疼痛干预: 骨质疏松性骨折术前和术后会出现明显疼痛, 缓解疼痛十分重要, 应尽早使用非甾体类药物 (non-steroidal antiinflammatory drugs, NSAIDs), 建议选用选择性环氧酶 2 (cyclooxygenase-2, COX-2) 抑制剂; 或降钙素 (calcitonin CT) (鲑鱼降钙素、依降钙素) 治疗; 镇痛效果不满意时可使用阿片类药物, 部分患者可应用多模式镇痛方法^[15-17,20,66,74,78-81]。

骨质疏松性骨折围手术期急性骨丢失干预: 骨质疏松患者骨折后即刻发生急性骨丢失, 而制动将进一步加剧急性骨丢失。应尽早使用降钙素治疗以缓解疼痛、减少急性骨丢失^[15-17,20,66,82]。

骨质疏松性骨折围手术期功能训练: 骨质疏松性骨折固定满意后, 应指导并鼓励患者积极开展早期功能锻炼, 避免术后骨量进一步丢失; 术后早期平卧位时, 可积极开展肢体肌肉等长收缩、关节被动活动; 手术创伤反应减轻时, 可在镇痛措施下增加肢体活动量; 对于椎体骨折固定稳定、髌部骨折固定稳定患者, 在医生 (或康复师) 指导下, 借助器具尽早开展短时间站立训练, 逐步开展短时间行走训练; 术后早期物理治疗可减缓肢体因制动所致的骨量丢失^[15-16,20,72,74,83-84]。

骨质疏松性骨折围手术期抗骨质疏松治疗药物选择

骨质疏松性骨折的病理基础是骨质疏松, 在围手术期应积极开展规范的抗骨质疏松药物治疗, 阻止骨质疏松进一步发展、防止再骨折发生。

基础治疗: 钙剂和维生素 D 是骨质疏松性骨折治疗的基础用药, 钙剂联合维生素 D 应用对治疗骨质疏松、促进骨折愈合十分必要^[20,85-88]。钙剂补充可改善骨矿化、减缓骨量丢失; 钙剂选择应注重元素钙含量, 骨折后推荐元素钙补充剂量为 1 000 mg/d。维生素 D 可促进钙在肠道吸收, 有利于骨基质矿化、抑制骨吸收, 减少再骨折发生; 维生素 D 补充剂量推荐 800 IU/d。

另外, 使用活性维生素 D (骨化三醇 0.25~0.5 μg/d、阿法骨化醇 0.5~1.0 μg/d) 可能增加肌力、改善平衡能力、降低跌倒风险、减少再骨折发生^[9,15,20,89-90]。

抗骨质疏松治疗: 应合理选择抗骨质疏松药物。

(1) 双膦酸盐: 双膦酸盐具有抑制破骨细胞介导的骨吸收功能、降低骨转换率、间接增加骨量的作用。口服双膦酸盐和静脉使用双膦酸盐均可显著降低绝经后骨质疏松再骨折风险^[9,15-17,20,66]。

骨质疏松性骨折后应用双膦酸盐时应明确患者肾功能状态, 血清肾小球肌酐清除率 (creatinine clearance, CCR) 低于 35 mL/min 时, 不建议应用; 应用双膦酸盐时联合“钙剂+维生素 D”治疗, 可提高疗效、降低药物不良反应发生率^[16,20,66,91-95]; 75 岁以上女性骨质疏松性骨折后, 如没有 BMD 检测结果, 可推荐应用口服双膦酸盐^[15,96-98]; 骨质疏松性骨折患者如有以下情况: 需要平卧者、口服用药依从性较差者、有反流性食管炎或食管疾病者、口服用药胃肠道反应较大者, 推荐选择静脉双膦酸盐治疗^[8,15-17,20,24]。

(2) 雌激素和选择性雌激素受体调节剂: 雌激素和选择性雌激素受体调节剂 (selective estrogen receptor modulators, SERMs) 具有抑制骨转换、阻止骨量丢失、改善 BMD 的作用。绝经后骨质疏松性骨折患者, 雌激素水平低且伴有更年期症状时, 推荐在专科医生指导下个性化应用雌激素或 SERMs^[15,66,98-100], 但不推荐在骨折急性期使用此类药物。

(3) 甲状旁腺激素类似物: 重组人体甲状旁腺激素片段 1-34 (recombinant human parathyroid hormone 1-34, rhPTH1-34,) 具有促进骨形成、改善骨重建作用, 可有效增加腰椎 BMD, 降低椎体和非椎体再骨折风险。有下列情况推荐应用 PTH1-34: 绝经后骨质疏松多发性骨折、双膦酸盐治疗后仍发生骨质疏松性骨折; 严重骨质疏松骨折 (T 值 < -3.0) 或多发骨质疏松骨折患者推荐使用甲状旁腺激素类似物^[15-16,20,66,98,100-105]。

(4) 维生素 K: 维生素 K 具有促进骨形成、抑

制骨吸收、提高骨量作用。维生素 K2 (45 mg/d) 联合钙剂和维生素 D 应用, 可降低骨质疏松性再骨折风险^[66,106]。

(5) 中药制剂: 中医中药在治疗骨质疏松及其骨折方面已有广泛的应用^[15,66,107-109], 含有人工虎骨粉、骨碎补总黄酮、淫羊藿苷等成分的中成药在治疗骨质疏松性椎体压缩性骨折、骨质疏松性股骨粗隆间骨折和骨质疏松性肱骨外科颈骨折时, 有缓解疼痛、缩短骨折愈合时间和改善 BMD 等临床疗效^[109-111]。

骨质疏松性骨折围手术期抗骨质疏松药物干预的特殊性

抗骨质疏松药物使用时间: 骨质疏松性骨折发生后, 抗骨质疏松药物应与骨折外科干预同时进行; 如果骨质疏松性骨折发生前已经接受抗骨质疏松药物治疗, 骨折愈合期间不建议停药^[16,20,112-113]。

应用口服双膦酸盐类药物时需空腹, 保持坐位或直立位 30 min; 若骨折后患者无法保持坐位或直立位, 建议选用静脉用双膦酸盐。双膦酸盐静脉应用时, 由于少数患者可能会出现一过性发热等药物反应, 建议于骨折术后数日应用, 避免与骨科手术引起的发热混淆^[113]。

抗骨质疏松治疗药物对内植物的影响: 抗骨质疏松药物治疗对骨质疏松性骨折手术植入物的影响, 尚未有大样本循证医学研究。现有研究表明, 骨质疏松性骨折内固定术后, 应用双膦酸盐可减少内固定周围骨量丢失、降低内固定松动发生率, 提高内固定手术疗效; 骨质疏松性骨折人工关节假体置换术后, 应用双膦酸盐可减少假体周围骨量丢失、降低假体松动发生率, 提高假体置换手术疗效^[15,20,114]。

骨质疏松性椎体骨折椎弓根内固定手术后, 应用 PTH1-34 可提高椎体骨量和骨质量、增加“骨-螺钉”界面把持力、降低椎弓根螺钉松动发生率, 提高椎体内固定手术疗效^[103]。

抗骨质疏松药物对骨折愈合的影响: 骨质疏松性骨折后, 早期应用钙剂和维生素 D (元素钙 1 000 mg/d、维生素 D 800 IU/d) 治疗可增加骨

痂面积、促进骨折愈合^[87-89]; 骨质疏松性骨折后, 无禁忌证时, 早期应用常规剂量的双膦酸盐治疗对骨折愈合无负面影响^[115-116]。

临床观察提示, 骨质疏松性髌部骨折内固定术后 1 个月应用 PTH1-34, 可促进骨折区骨痂形成; 骨质疏松性桡骨远端骨折 10 d 内应用 PTH1-34, 骨折愈合时间显著缩短^[117-118]。

骨质疏松性骨折术后管理

骨质疏松性骨折不仅仅是一种病理性骨折, 更具危害的是首次骨折一旦发生, 再骨折的风险急剧上升, 故在围手术期就应启动再骨折的风险评估和干预^[16,20]。

骨质疏松性骨折术后再骨折危险因素: 包括 BMD 低、高龄等全身因素; 脊柱骨折、下肢负重部位骨折等局部因素。跌倒是独立于 BMD 以外导致骨质疏松再骨折的危险因素^[9,15-16,20,66,83,100]。

骨质疏松骨折术后再骨折预防: 骨质疏松性骨折术后, 缩短“平卧位转换站立位”时间^[119]、增加防跌倒措施^[2,24]、早日出院、提高有效家庭康复训练, 有助降低骨质疏松性骨折术后再骨折的发生率^[120]。

骨质疏松性骨折术后的常规管理: 骨质疏松性骨折术后管理是防治骨质疏松再骨折的重要措施^[15-16,20]。骨质疏松性骨折术后管理主要分为两部分: 骨折术后管理和抗骨质疏松治疗管理; 管理目标是促进骨折愈合, 提高骨质疏松及其骨折治疗的依从性和有效性^[83]。

骨质疏松性骨折术后管理: 仔细了解患者骨骼功能恢复状况, 根据骨折类型和治疗方案确定骨折术后预期功能目标; 采用相应骨科措施, 预防骨折后并发症, 定期评估骨折愈合, 帮助骨折愈合后功能康复^[16,20,83,121-123]。

骨质疏松性骨折术后抗骨质疏松治疗管理: 骨质疏松性骨折术后抗骨质疏松治疗管理包括向患者说明药物治疗依从性与骨折治疗疗效的相关意义^[16,20,83]; 规范用药、定期监测; 对抗骨质疏松治疗患者推荐每 3 个月或每 6 个月检测 1 次骨转换指标、12 个月检测 1 次 BMD, 以评价药物治疗效果^[66,120,124]; 双膦酸盐

类药物一个治疗周期通常为3~5年(口服药物5年,静脉药物3年),PTH1-34治疗周期不超过24个月^[66,102-104,100,125-126]。

双膦酸盐类药物治疗3~5年后,如果满足以下条件可考虑暂停用药:治疗期间未发生再骨折;无新的骨折风险因素增加;无显著BMD下降;严重骨质疏松性骨折患者股骨颈BMD T值提高。停药后应每半年检测骨转换指标、每年检测BMD,以确定是否需要再次进行药物治疗^[2,66,100]。

骨质疏松性骨折后专项管理:骨质疏松性骨折后,推荐接受骨折联络服务(fracture liaison service, FLS); FLS机制的核心是医院增设一个骨质疏松骨折协调员(或者是机构),主要工作内容包括骨折患者的骨质疏松诊断、骨质疏松骨折患者评估和个性化治疗、治疗过程的随访、康复和防跌倒管理^[16,20,66,83,127-132]。

另外,骨质疏松骨折患者的医患教育、监护人教育、专项医患沟通(doctor-patient communication, DpC)机制,均可提高骨质疏松性骨折后管理综合疗效,切实降低骨质疏松性再骨折的发生率^[83,133-134]。

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