

假体周围骨吸收的机制及防治进展

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中图分类号: R454 文献标识码: A 文章编号: 1006-7108(2011)02-0162-05

摘要: 关节置换术目前已经在临床广泛的应用,但是术后 10 年翻修率达到 10%,其主要原因是假体松动。从影像学资料分析,松动假体的周围存在大量的骨质吸收,本文通过对近 10 年的相关文献的回顾,分析了假体周围骨质吸收的机制以及相应的预防治疗思路。

关键词: 关节; 假体; 骨质吸收

Progress of the mechanism and the prevention and treatment of bone resorption around the prosthesis BAO Hangxing, WANG Jinfa, CAI Yunhuo, et al. Zhejiang University of Traditional Chinese Medicine, Hangzhou 310006, China

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Abstract: Joint replacement surgery is now widely used in the clinic, however the renovation rate exceeds 10% 10 years after the operation. The main reason is the prosthesis loosening. According to the imaging data analysis, there is a lot of bone resorption around the loosening prosthesis. Based on a review of the literature in the past decade, we analyze the mechanism of periprosthetic bone resorption and provide the corresponding preventive treatment ideas.

Key words: Joint; Prosthesis; Bone resorption

关节置换术目前已经在临床广泛的应用,全球每年有 100 万关节置换,而 10 年后翻修率达 10%^[1]。长期跟踪调查显示,术后病人假体生存率达到 20 年的在 80% 以上^[2]。因此,关节置换术应用于更多年轻以及运动量大的个体中^[3]。但是,有大量的因素导致关节置换的远期效果不佳,主要是无菌性骨质吸收、外科手术技术、术后微骨折等。假体周围骨质疏松以及后期的假体松动都和慢性的无菌性炎症相关。对于假体周围骨吸收的原因,近年来,大家比较公认的因素就是假体磨损颗粒,假体的使用寿命往往和磨损颗粒引起的骨质吸收有关^[4],我们搜索了近 10 年的国内外文献综述如下。

1 机制研究

关于假体周围骨吸收的机制,是一个复杂的生理病理过程。许多细胞活素不同程度的影响着骨质吸收的过程,如 CSF-1、IL-1、TNF- α 、IL-6 等^[2-5],但是,随着 RANK/RANKL 路径的发现,以及对它作用

机制的研究,对于磨损颗粒引起的假体周围骨吸收的认识,已经得到了长足的发展。破骨细胞分化成熟的过程中,有 3 个非常重要的因子,即骨保护素(OPG)、细胞核因子 kappaB 受体活化因子(RANK)、细胞核因子 kappaB 受体活化因子配基(RANKL),它们都是肿瘤坏死因子配体和受体家族成员,这 3 个因子所形成 OPG、RANKL、RANK 系统介导了 OC 形成、分化过程中所必须的细胞间信号传递。当刺激因素作用于成骨细胞(osteoblast, OB)、基质细胞(stromal cell, SC),诱导其膜上表达 RANKL 分子,通过与 OC 前体细胞膜上 RANK 直接结合,将信号传入前体细胞,引起级联瀑布反应,使 OC 分化成熟;而 OPG 则由 OB、SC 旁分泌发挥作用,竞争性与 RANKL 结合,封闭 RANKL 与 RANK 的结合,将抑制 OC 的分化、成熟。RANKL 是可直接诱导 OC 分化的细胞因子,其他细胞因子或激素都是直接或间接地通过 RANKL 与 RANK 或 OPG 的相互作用来影响 OC 分化与成熟的。磨损颗粒,正向调节 T 淋巴细胞和巨噬细胞,通过 RANKL 途径,促进破骨细胞的分化^[6],从而引起假体周围骨吸收。

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RANK/RANKL 路径是聚乙烯颗粒病的最后一步,但在松动假体周围的假模中分离出了大量增殖的细胞活素,包括 TNF- α 、IL-1、IL-3、IL-6、TGF- β 、PGE2、CSF-1 等^[7-8]。我们将讨论其中几种的作用机制。

1.1 TNF- α

TNF- α 已经被证实是引起聚乙烯磨损颗粒导致骨吸收的一个重要的破骨细胞因子^[9-10]。它能够调节其他炎症因子的作用,比如 IL-1, IL-6, IL-8, 和 GM-CSF 等^[10-12]。TNF- α 能够和 RANKL 在假体周围骨质吸收中发生协同作用,同时能够独立诱导 TNF- α 受体 NF- κ B 被激活^[13]。Christopher 等^[14]通过实验证明 TNF- α 通过激活 NF-B 从而导致假体周围骨吸收。将 11 个翻修术病人假体周围骨吸收部位的组织进行病理切片免疫组化,同时对 10 个初次关节置换病人滑囊组织进行同样处理后发现,翻修组中 RANK、RANKL、TNF- α 强烈表达,对照组中表达较少。统计学分析后发现,TNF- α 和 RANKL 在引发假体周围骨吸收的过程中有协同作用。

1.2 IL-1(白细胞介素-1)

IL-1 是一个炎症前因子,可以引起发热,血管粘附因子的活化,引发关节炎等。IL-1 也是通过激活 NF- κ B 从而引发炎症。IL-1 是破骨细胞因子促进 RANKL 的表达,从而导致假体周围骨吸收^[15],TNF- α 也是通过 IL-1 作为媒介。IL-1 可以直接促进单核破骨细胞前体的增值和分化,但需要 RANKL 的协同作用^[13-15],因此可以理解,IL-1 的作用是 TNF- α 引导的假体周围骨吸收的下游环节,促进 RANKL 的表达,以及直接激活破骨前体细胞。

1.3 IL-3(白细胞介素-3)

IL-3 对 OC、OB 的分化都具有重要影响。骨髓瘤患者血浆中 IL-3 比正常水平高,经过研究发现 IL-3 可加强 OC 的分化形成^[16]。但是,研究也发现 IL-3 可抑制 OC 前体的分化过程,它抑制 RANKL 的 NF-kappa 信号通路,而且也可通过下调 TNF 受体来抑制 TNF-Dpha 对 OC 的诱导过程,对成熟 OC 没有抑制作用,IL-3 还可间接抑制 OB 的形成^[17]。这些似乎矛盾的研究结果提示 IL-3 在 OC 分化成熟的过程中起着复杂的调节作用,以维持骨代谢的平衡。

1.4 IL-6(白细胞介素-6)

IL-6 属于 gp130 细胞因子家族,多种细胞均可分泌。骨组织中的 IL-6 主要来源于 OB SC 分泌,PKG 可促使 OB 表达 IL-6^[18]。IL-6 与 IL-3 协同支持造血干细胞、OC 前体的生长;IL-6 也能增强其他

细胞因子或激素对 OC 的作用,如增强 PTHrp 维持体内钙平衡和调节 OC 骨吸收的作用。IL-1 和 TNF- α 是具有刺激骨吸收作用的细胞因子,主要是它们能诱导 OB 产生 IL-6,IL-6 可与 PGE2 协同作用通过 OPG/RANK/RANKL 系统来调节 OC 的分化^[19]。

1.5 前列腺素(Prostaglandin, PGE)

骨组织中的 PGE2 主要由 OB 分泌,是强有力的骨形成和骨吸收刺激因子,对 OC 的分化具有双重作用,低浓度的 PGE2 可诱导 OC 的分化,高浓度的 PGE2 对分离的 OC 有抑制作用。PGE2 介导多种细胞因子引起的骨吸收^[20],研究表明,PGE 呈剂量依赖性促进小鼠 OB 胞内 cAMP 合成,并通过 cAMP-PKA-CREB 信号传导途径,促进 OB 骨基质金属蛋白酶-1 (matrix metalloproteinase-1, MMP-1) 与 IL-1G 基因的表达,促进 OB 合成和分泌 MMP-1 与 IL-1G,从而有效抑制骨吸收。在 RANK/RANKL、OPG 系统的 OC 分化过程中,PGE 与 IL-6 的相互影响起着重要作用^[21]。PGE2 还可通过诱导 PKC-ERK 蛋白激酶信号传导途径中 ERK 激酶的激活,介导 TGF- β 促进 OB 增殖的效应。

尽管大量的炎症因子已经被发现,但是,没有一种被证实是在假体周围骨吸收中发挥着决定性的作用。因为在阻断某一个炎症因子后,假体周围的无菌性炎症并没有中断。实验已经证明 RANKL-RANK-OPG 的相互作用在导致假体周围骨吸收中发挥了关键作用。成骨细胞与破骨细胞之间存在相互调控机制,成骨细胞或骨髓基质细胞表达的核因子- κ B 受体活化因子配体 (receptor activator of NF- κ B ligand, RANKL) 和巨噬细胞集落刺激因子 (macrophage colony stimulating factor, M-CSF) 与破骨细胞的形成有关,成骨细胞表面的 RANKL 与破骨细胞前体细胞表面的 RANK 结合后,引发一系列反应,促使破骨细胞前体细胞分化和成熟为有功能的破骨细胞,抑制破骨细胞的凋亡。成骨细胞及骨髓基质细胞同时又分泌表达骨保护素 (osteoprotegerin, OPG),与 RANKL 竞争性结合,阻止 RANKL 与 RANK 之间的结合,防止骨的过度吸收。因此,RANKL 与 OPG 的比值是维持局部骨代谢平衡的关键。钛离子可能使破骨细胞与成骨细胞之间的联系通讯遭到破坏,直接作用于破骨前体细胞并且使其与成骨衍生因子如 M-CSF 和 RANKL 失去联系,或者是破坏了 RANK 与 OPG 之间的平衡,从而导致成骨与破骨失调,引起骨质吸收。^[22]

2 假体周围骨吸收的治疗和预防思路

2.1 抗 TNF- α 的治疗

TNF- α 在造成假体周围骨吸收的无菌性炎症中所发挥着重要的作用,针对 TNF- α 的治疗是否能够治疗或者缓解假体周围骨质吸收?这个发现为我们的临床治疗提供了一个很好的思路。依那西普是一种 TNF- α 拮抗剂,用于治疗类风湿关节炎。动物实验证明 TNF- α 可以抑制破骨细胞造成的骨吸收^[23]。然而一个 20 个患者组成的临床观察显示,使用依那西普组和安慰剂组在抑制假体周围骨吸收方面没有明显差别^[24]。但这只是初步的临床观察,还不足以对依那西普针对假体周围骨吸收的治疗作用做出评估。

2.2 抗炎治疗

假体周围骨吸收是由于无菌性炎症引起的,那么抗炎治疗就必不可少。这是一种比抗 TNF- α 治疗更为广谱的抗炎治疗,这种广谱的治疗是非常必要的,因为无菌性炎症时一种非常复杂的过程,其机制尚未清楚。Im 等^[27]进行的一次体外实验证明,IL-4, IL-10 可以明显抑制 TNF- α 和 IL-6 的表达。然而,目前还没有人体实验的报告。Weiping Ren 等^[26]将红霉素进行体外实验证实红霉素可以抑制破骨细胞的产生和分化,从而抑制骨吸收。

2.3 假体材料的改进

选择一种耐磨,并且具备合适强度的假体材料对于假体寿命有着决定性的意义。

从 1971 年开始,人们开始采用 Al_2O_3 陶瓷替代原有的金属材料,到目前为止已经经历了四代产品。和传统的金属材料相比, Al_2O_3 陶瓷具有高强度和硬度,因此可以减少研磨颗粒的产生,从而提高假体寿命。大量的实验和临床研究也证明了这一点^[27]。Donatella Granchi 等^[30]的研究表明, Al_2O_3 陶瓷磨损颗粒所引起的破骨细胞分化明显低于高分子聚乙烯颗粒,并且在分子水平证实了这个观点。高分子聚乙烯颗粒改变了人类成骨细胞所释放的溶解因子的结构,促进了破骨前体细胞 c-src 的表达,由此证明聚乙烯颗粒引起破骨细胞的计划和粘附,从而导致骨吸收。相反, Al_2O_3 陶瓷磨损颗粒降低了 c-fos 的表达,说明它降低了巨噬细胞向破骨细胞的分化。Germain 等^[29]的体外模拟研磨实验表明, Al_2O_3 陶瓷和聚乙烯的磨损率要低于金属和聚乙烯的磨损率,同时,后者所产生的研磨颗粒的毒性也要高于前者。Urban 等^[32]报道 Al_2O_3 陶瓷对聚乙烯的年磨损率为

0.034 mm;Hernigou^[33]报道 Al_2O_3 陶瓷对聚乙烯的年磨损 0.071 mm。

ZrO_2 陶瓷是另一种替代金属的假体材料,和 Al_2O_3 陶瓷相比,它的强度更高,硬度高好,更加不易变形,因此用 ZrO_2 陶瓷设计的髋关节假体,头更小,颈更长,且不会增加再骨折率,但是,临床医生更愿意选择 Al_2O_3 陶瓷假体^[32-34]。因为 ZrO_2 陶瓷假体在 10 年内的变形率更高,产生更多的研磨颗粒,最终导致假体松动和再骨折。而 Al_2O_3 陶瓷假体的生存率高于 ZrO_2 陶瓷假体,随着材料学的发展,新一代的 Al_2O_3 陶瓷假体将会有更优异的表现。

2.4 中医药治疗

祖国医学博大精深,对于防治假体周围骨质吸收,延长假体寿命,我们可以从中寻求一定的防治思路。假体周围骨吸收,其本质是手术部位局部反应所导致的骨质疏松,因此,可以借鉴已有的中医药治疗骨质疏松的经验,应用于关节置换术后的病人。

2.5 手术技术的注意点

(1)处理髓臼时要尽量增加髓臼底部的深度,从而减少股骨头中心至躯干中线杠杆臂距离,增长股骨头中心至大转子的杠杆臂,达到减轻髓关节假体受力的目的。(2)准备髓臼时,外上缘不要磨锉过多,否则可导致人工髓臼不稳,造成松动。(3)人工髓臼杯置入时,角度要与躯干纵轴 45°角。(4)选择的人工股骨头柄要与髓腔相匹配,必要时可用髓腔绞刀扩大,以使人工股骨头顺利插入,不致造成股骨近端劈裂。(5)如人工股骨头柄杆要用骨水泥固定,则骨水泥应均匀包绕在柄杆周围,不要集中在髓腔上部,插入时不要摆动,否则骨水泥轨迹增大,会影响假体的稳定性^[35]。

3 总结

假体周围骨质吸收,以及它所带来的假体松动,是影响关节置换术后疗效的关键因素,若能克服这一难题,将会大大提高关节置换术的疗效。目前认为对于假体周围骨吸收主要是磨损颗粒导致的无菌性炎症所导致的,也发现了很多相关的炎症因子,但是最终,最为确定的还是 RANK-RANKL-OPG 途径。目前对于假体周围骨吸收的认识,还不是非常明确,有待我们进一步研究。在明确其机制的基础上,找到治疗的方法。随着材料学的发展,以及手术技术的提高,关节置换术将会得到长足的发展。同时,我们也要积极运用中医药,防治假体周围骨吸收。在多种途径的共同努力下,必将为关节疾病患者带来

更好的生活质量。

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(收稿日期: 2010-08-02)

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(收稿日期: 2010-09-08)

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英文刊名: CHINESE JOURNAL OF OSTEOPOROSIS
年, 卷(期): 2011, 17 (2)

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