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·综述·

# 侵袭性牙颈部外吸收的研究进展

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【摘要】 牙体吸收可分为生理性吸收和病理性吸收。成熟乳牙的牙根吸收即为生理性吸收。病理性牙体吸收则包括牙内吸收和牙外吸收。牙内吸收也被称为髓腔内吸收，包括炎症性吸收和替代性吸收。牙外吸收始发于牙根外表面或牙颈部，可分为炎性吸收、替代性吸收、压力性吸收和侵袭性颈部外吸收等。侵袭性颈部外吸收是一种由多因素导致牙体硬组织丢失的病理性损害，通常从釉牙骨质界开始，在牙本质中周向或水平方向延伸，大多不侵犯牙髓。正畸、创伤、漂白、全身性疾病及某些药物的使用等均可导致侵袭性颈部外吸收。其临床表现通常为无症状或症状不明显，大部分均是在影像学检查时发现。因为在根尖平片影像中往往会误诊为龋病或牙内吸收，所以为了更好地了解牙齿颈部外吸收的情况，临床上使用锥形束CT（cone beam CT，CBCT）进行检查。对侵袭性牙颈部外吸收的发病机制及病因尚未完全了解，会导致临床上对该疾病的疏忽或治疗不当，甚至造成牙齿丧失。本文就侵袭性牙颈部外吸收的组织病理学表现、发病机制、易感因素、诊断和治疗等方面进行综述。

【关键词】 牙吸收；　侵袭性颈部外吸收；　核因子κB受体活化因子；　核因子κB受体活化因子配体；　骨保护素；　炎症因子；　多发性特发性颈部外吸收；　易感因素；　锥形束CT
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**Research progress on invasive cervical resorption**

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【Abstract】 Tooth absorption can be divided into physiological absorption and pathological absorption. Root absorption of mature deciduous teeth is physiological absorption. Pathological absorption includes internal absorption and external absorption. Internal absorption, also known as intramedullary absorption, includes inflammatory absorption and alternative absorption. External tooth absorption originates from the outer surface of the root or the neck of the tooth and can be divided into inflammatory absorption, alternative absorption, pressure resorption and invasive cervical resorption. Invasive cervical resorption (ICR) is pathological damage caused by many factors, which usually begins in the cementoenamel junction and extends peripherally or horizontally in the dentin. It hardly invades the pulp. Orthodontic devices, trauma, bleaching, systemic diseases, and the use of certain medications can all lead to invasive cervical resorption. The clinical manifestations of ICR are usually asymptomatic or not obvious, and most of which are found in imaging examinations. Because caries and internal absorption are often misdiagnosed through plain apical radiography, cone beam computed tomography (CBCT) can help to better understand the situation of invasive cervical resorption. Because the pathogenesis and etiology of invasive cervical resorption are not fully understood, clinical negligence and inadequate treatment of invasive cervical resorption can even cause unnecessary tooth loss. This article reviews the latest research progress on the histopathologic features, pathogenic mechanism, susceptibility factors, diagnosis and treatment of ICR, with special emphasis on susceptibility factors and their mechanisms.

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【Key words】 tooth resorption; invasive cervical resorption; receptor activator of nuclear factor; receptor activator for nuclear factor-κB ligand; osteoprotegerin; inflammatory factor; multiple idiopathic external cervical root resorption; susceptibility factors; cone beam CT
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侵袭性牙颈部外吸收(invasive cervical resorption,ICR)是一种特殊的牙外吸收。由于各种因素导致釉牙骨质界处上皮下牙体硬组织的受累或缺失[1]。ICR发病率为1.35%,可发生在各年龄段,男性的发病率高于女性[2],多发生在前牙区,上颌和下颌中切牙多见[3]。其病因尚不明确,局部刺激、全身性疾病和某些药物的使用均与ICR的发生有关。ICR是一个动态且复杂的过程,临床表现通常无症状,可能会出现刷牙不适感、探诊出血、牙颈部粉红色或红色改变以及病变晚期引起的牙髓症状[4],大部分是从影像学检查中发现。随着锥形束CT(cone beam CT,CBCT)在口腔领域的使用,ICR的诊断也包括了三维分类,诊断更加精准,对临床的治疗方法给予了很大的指导作用,从而使牙齿的预后更佳[5]。本文就ICR的组织病理学表现、病因机制、易感因素、诊断和治疗方面的最新研究进展作一综述。

## 1　ICR的组织病理学表现

ICR在组织病理学表现上分为3个阶段,即入口阶段、吸收阶段及修复阶段[6]。第一阶段:入口阶段起始的标志是牙周膜的剥离和保护性牙骨质的破坏[7]。发生ICR的活髓牙和根管治疗后的死髓牙在入口阶段表现不同。活髓牙在吸收入口区域可以有3种表现,即生成新的牙骨质和牙周膜或者发生强直反应,第三种情况是发生进一步的吸收。根管治疗过后的牙齿因为没有牙髓的营养,所以不会形成新的牙骨质和牙周膜并且吸收速度更快。第二阶段:吸收阶段则表现为病变吸收部位充满高度血管化的纤维结缔组织以及大量的炎性细胞,同时可观察到吸收部位的细胞胞质中含有丰富的溶酶体,溶酶体可吸收脱矿的牙本质和牙釉质基质[8]。第三阶段:修复阶段在吸收陷窝处有矿物质沉积,可见到修复性骨组织逐渐形成,该阶段往往是修复和吸收交替进行。

## 2　ICR的发病机制

牙体硬组织吸收与核因子κB受体活化因子配体(receptor activator for nuclear factor-κB ligand,RANKL)-核因子κB受体活化因子(receptor activator of nuclear factor-κB,RANK)-骨保护素(osteoprotegerin,OPG)信号通路相关。多种炎症因子上调RANKL从而促进破骨细胞形成,OPG与RANKL竞争性结合,抑制破骨细胞形成,二者的比值影响吸收[9]。目前ICR的确切发病机制仍不清楚,有学者研究发现ICR的发生可能同样是RANKL与OPG二者之间的比值失衡导致[10]。除此之外,有学者从基因水平研究发现靶基因miR-122-5p在肉芽肿性组织中表达上调[11]、干扰素调节因子8基因(IRF8G388S)发生突变[12]会引起炎症反应和吸收,这些研究结果可为进一步研究ICR的发病机制奠定基础。

## 3　ICR的易感因素

### 3.1　局部因素

引起ICR的局部刺激因素很多,例如创伤、牙齿漂白、正畸、牙周手术和不良口腔习惯等[13]。牙外伤多发生于年轻患者,牙齿创伤后会引起牙外吸收[14]。牙震荡是牙齿支持组织的损伤,牙齿有明显的叩诊不适,但没有异常松动或移位。牙齿发生牙震荡后釉牙骨质界处的连接受影响,该部分暴露后可能产生一些不被机体识别的蛋白质,在机体免疫系统识别并清除这些蛋白质的过程中引发ICR[15]。牙震荡后可能还会伴发牙根吸收的后遗症[16]。

临床上对于各种原因导致的牙齿变色问题口腔医生通常利用漂白的方式解决[17]。一项系统性综述表明漂白剂(如过氧化碳酰胺、过氧化氢及过硼酸钠)针对牙齿变色的漂白效果虽然较好[18],但是发现牙齿漂白剂与侵袭性颈部外吸收存在相关性[19]。漂白剂可以通过牙本质渗透至牙周膜,继发的炎症反应可导致吸收。除此之外,漂白剂产生的酸性环境也会增加破骨细胞的活性,从而导致多发性特发性颈部外吸收(multiple idiopathic external cervical root resorption,MIECRR)。当3颗以上牙齿出现侵袭性颈部外吸收时称为MIECRR,往

往不易察觉且进展迅速[20]。MIECRR好发于年轻女性患者，可能与激素相关，临床表现几乎无症状，影像学检查往往表现为边缘呈扇形的放射状透明带，病变往往始于牙颈部的近中或远中，进而进展整个颈部，严重则向牙髓延伸，但大部分不侵犯牙髓[21]。

Brito等[22]关于正畸过程中上颌中切牙发生侵袭性吸收的病例报告中，认为ICR是正畸治疗过程中的并发症之一。Khalaf等[23]研究表明正畸和ICR存在显著相关性，正畸过程中正畸力过大时压力侧可能会刺激破牙细胞，使牙齿发生吸收。当手术辅助技术结合正畸力学产生的持续力一起使用时，可能会增加ICR发生的风险，因此在正畸过程中应谨慎使用这些技术以及仔细监测牙齿ICR的发生[24]。拔牙及牙周手术均可破坏釉牙骨质界，使该处牙周膜断裂以及牙骨质丢失，最终导致侵袭性颈部外吸收[25]。

### 3.2　全身因素

虽然至今仍没有证据表明某些全身疾病与ICR的发生存在直接联系，但是一些未受过局部刺激因素影响的患者也发生了ICR，猜测可能与某些全身疾病相关。

乳糜泻引起的维生素$D_3$水平的降低可能是引发多发性颈部外吸收的一个相关因素[26]。原因可能是小肠的慢性炎症会影响脂溶性维生素的吸收，特别是维生素$D_3$，进而导致血液中甲状旁腺激素水平的升高，甲状旁腺激素分泌的增加可直接影响成骨细胞和破骨细胞的形成[27]。

硬皮病（systemic sclerosis，SSC）是免疫介导的疾病，早期表现为皮肤紧绷，偶尔有三叉神经痛或舌咽神经痛的症状。也有学者研究表明SSC会引发一些口腔表现，例如口干、关节紊乱及侵袭性颈部外吸收[28]。硬皮病引起ICR的原因可能是皮肤挛缩引起局部血供减少导致局部区域微循环障碍，进而发生的缺氧情况增强了破骨细胞的活性，最终导致吸收[29]。

有学者研究发现ICR病变和周围牙齿组织内存在着梯度缺氧[30]。ICR影像学上髓石的表现同时也表明了牙髓内低氧环境的存在。有学者研究发现糖尿病是ICR发生的易感因素之一[6]。二者之间的潜在联系可能是低氧的存在。糖尿病患者的低氧微环境会引发氧化应激和炎症反应，同时低氧环境下破骨细胞被激活，启动骨吸收，引发ICR。

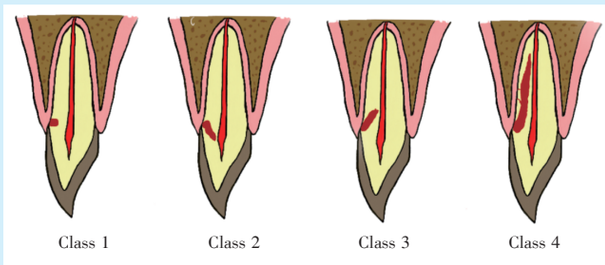
髓鞘少突胶质细胞糖蛋白（myelin oligodendrocyte glycoprotein，MOG）抗体相关疾病是一种罕见的自身免疫类疾病。一例关于MOG抗体相关疾病患者口腔经过CBCT检查后发现颈部外有吸收，推测颈部外吸收可能与MOG抗体相关疾病中炎症因子的增多有关。虽然无法确认其机制，但是临床上针对该类患者出现ICR后应密切观察并积极治疗[31]。

### 3.3　药物因素

根据一些临床病例文献可以得知某些治疗骨质疏松药物的使用与颈部外吸收也存在相关性。随着年龄的增长，骨质疏松症受到越来越多的重视，针对治疗骨质疏松症的药物也在不断增多。一位服用双膦酸盐（bisphosphonate，BPS）的骨质疏松患者发生了ICR，研究发现BPS具有促炎特性，通过影响炎症细胞因子的释放来影响ICR的启动和进展[32]。治疗骨质疏松的另一药物地诺单抗（Denosumab）是一种全人单克隆抗体，对RANKL具有高度亲和力，可以抑制破骨细胞的产生，从而降低骨转换。病例报告表明地诺单抗与ICR的发生存在相关性，即停用地诺单抗后，骨密度快速下降，骨转换标记物急剧增加，ICR加速进展[33]。另一项研究发现患者服用地诺单抗期间发生了ICR，这可能意味着不依赖RANKL的破骨细胞形成途径也参与了ICR的发生[34]。因此对于服用该药物的口腔患者来说，在开展侵入性牙科治疗前后需要谨慎检查，并与主治医师沟通后决定。

## 4　ICR的诊断

因为ICR大多数无临床症状，所以影像学检查对于ICR的诊断和治疗具有重要意义。ICR的影像学分类是由Heithersay[13]首次提出，通过根尖周成像将ICR分为4类（图1）：（1）Ⅰ类，颈部范围的小侵袭性病变，仅涉及牙本质浅层；（2）Ⅱ类，边界明确的侵袭性吸收病变，近冠髓，未延伸或很少延伸到根部牙本质；（3）Ⅲ类，范围更大的侵袭性吸收病变，不仅累及冠部牙本质，而且至少延伸至牙根的冠1/3；（4）Ⅳ类，病变范围已延伸超过牙根的冠1/3。Heithersay分类是第一个用于ICR病变进行分类的系统，旨在指导治疗并帮助确定患者预后。虽然Heithersay分类是从二维方向上定义的，但是因为其应用范围广泛所以大多数临床治疗指南仍基于该分类系统。在一项评估体内ICR病变体积与其分类和治疗方案的相关性研究中，Heithersay分类下的Ⅰ、Ⅱ类病变牙齿修复后的抗折能力更强，预后更好，而对于Ⅲ、Ⅳ类病变来说需要




Class 1: denotes a small invasive resorptive lesion near the cervical area withshallow penetration into dentin; Class 2: denotes a well-defined invasive resorptive lesion that has penetratedclose tothe coronal pulp chamber but shows little or no extension intothe radicular dentin; Class 3: denotes a deeper invasion of dentin by resorbing tissue, not onlyinvolving the coronal dentin but also extending at least to the coronalthird of the root; Class 4: denotes a large invasive resorptive process that has extended beyond the coronal third of the root canal

Figure 1　Schematic diagram of Heithersay classification[13]
**图1**　Heithersay分类[13]示意图

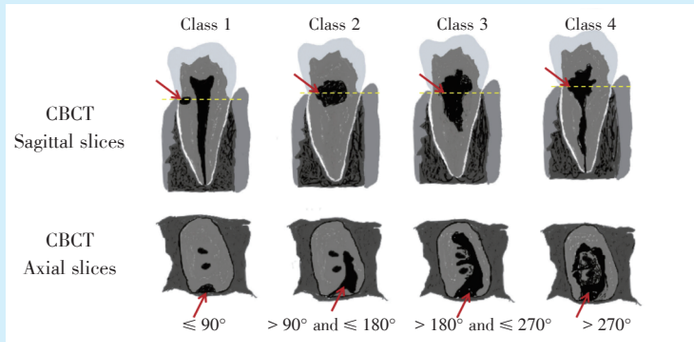
通过CBCT进行进一步的评估[35]。

随着CBCT在牙髓治疗方面的介入，与传统的根尖周成像相比，大量文献支持CBCT成像具有更高的准确性。在传统根尖片中，牙齿解剖结构和投射角度可能掩盖许多病理细节，CBCT不但可以消除这些影响因素，而且能够在病变早期阶段检测出牙根吸收的情况[6]。除此之外，CBCT成像还可以帮助临床医生更好地了解病变的大小、位置和范围以及更加清楚地识别患牙的牙髓和根尖与邻近重要结构的解剖关系[36]。从CBCT成像中可以观察到颈部外吸收分为三个阶段，即入口阶段、吸收阶段和修复阶段。ICR入口往往在牙齿的近远中釉牙骨质界且吸收往往达牙根的中1/3。故Patel等[5]从三维方向上对ICR进行了新的分类。Patel分类标准是从病变的高度、周向扩散的范围和与根管的距离这三个维度展开(图2)。而Rohde分类系统则从ICR引起的牙本质损失量入手，通过牙本质损失量与牙齿周长之间的关系进行分级(图3)[37]。虽然二者从不同的角度对ICR进行分类，但是在评估ICR病变体积的研究中均具有重要意义。目前临床上对于ICR病变体积的评估均是由影像学医生进行测量，这不仅要求影像学医生具有严格的操作技能，而且影像学医师的测量结果同时影响着临床医师对于ICR的治疗。

## 5　ICR的治疗

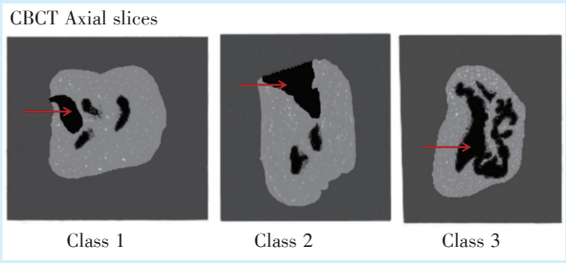
ICR的治疗包括手术方法、非手术方法、意向性再植、不可治疗的定期观察以及拔除[38]。可根据ICR病变的入口大小、根部的延伸范围以及涉及的牙本质量选择相应治疗手段[39]。

对于不侵犯牙髓和根尖周组织的Heithersay 1级和2级ICR病例来说，病变直视条件可见时可通过结合机械刮除和化学清创去除吸收组织[40]，使用玻璃离子或复合树脂进行充填；病变直视情况下不可见时，可在牙周翻瓣手术提供可视条件下，使用可再生牙骨质和牙周组织的MTA、硅酸钙基材料(如Biodentine)进行修补[41]。由于MTA固化时间长以及抗断裂性弱于Biodentine，因此Biodentine是修复ICR更好的选择[42]。值得注意的是前牙区ICR的治疗更加需要多学科联合治疗[43]。对于Heithersay 3级和4级的晚期ICR缺损来说，非手术方法中的活髓切断术、部分活髓切断术及根




Lesion height: 1: at the cementoenamel junction level; 2: extends into the coronal one third of the root; 3: extends into the mid one third of the root; 4: extends into the apical one third of the root. Circumferential spread: A: ≤90°; B: > 90°and ≤180°; C: > 180° and ≤270°; D: > 270°. Lesion depth: d: lesion confined to the dentin; p: lesion may invade the pulp of the tooth. Patel classification: Class 1: 1 & A & d; Class 2: 2 & B & d; Class 3: 3 & C & d; Class 4: 4 & D & p. Red arrow: lesion of invasive cervical resorption; yellow dotted line: alveolar ridge level

Figure 2　Schematic diagram of Patel classification[5]
**图2**　Patel分类[5]示意图




Red arrow: lesion of ICR; Class 1: significant dentin resorption in less than one third of the tooth circumference; Class 2: significant dentin resorption in less than one of the third tooth circumference with obvious perforation defects; Class 3: significant dentin resorption involving greater than one third of the tooth circumference

Figure 3　Schematic diagram of Rohde classification[37]

**图3**　Rohde分类[37]示意图

管治疗术也是一种可靠的方法，对于同时伴有穿孔的患牙，需要在外科手术结合显微镜辅助下进行修补。除此之外病变缺损较大的患牙也可通过意向性再植的方法，对于没有治疗价值的患牙，建议患者定期复诊观察，无法治疗的患牙选择拔除。

## 6　小　结

因为牙颈部外吸收可发生在任何牙位，任何年龄，并且具体确切病因仍然未知，所以对ICR的管理仍然是一个临床挑战，这就要求临床医生应该对ICR的可能性有高度认识，在临床和影像学评估的基础上，采取多学科的综合治疗方法。ICR的预后似乎高度依赖于病变的早期检测，因此要求临床医生更多的掌握ICR的相关知识，做到早发现，早诊断，早治疗，提高牙齿的预后。

**【Author contributions】** Liu QP wrote the article. Jiang Q conceptualized and revised the article. All authors read and approved the final manuscript as submitted.

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