

分化型甲状腺癌术后放射性碘治疗与甲状旁腺激素水平相关性研究进展

顾 硕¹, 王 飞², 李春雨³, 崔丽丽², 赵军玉^{2,4*}

¹济宁医学院临床医学院(附属医院), 山东 济宁

²山东第一医科大学第一附属医院(山东省千佛山医院)内分泌与代谢病科, 山东省风湿免疫病转化医学重点实验室, 山东省肾脏病研究所, 山东 济南

³济南(银丰)医院老年病科, 山东 济南

⁴山东大学齐鲁医学院山东省千佛山医院内分泌与代谢病科, 山东 济南

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摘 要

目的: 分化型甲状腺癌(differentiated thyroid cancers, DTC)术后常采用¹³¹I辅助清甲治疗, 甲状旁腺与甲状腺解剖毗邻, 放射性辐射或造成甲状旁腺损伤、甲状旁腺激素(parathyroid hormone, PTH)异常。本文系统梳理DTC术后¹³¹I治疗对甲状旁腺功能及PTH水平的影响、相关作用机制、危险因素与临床干预措施, 为临床安全开展放射性碘治疗提供参考。方法: 检索中英文数据库收录的分化型甲状腺癌、¹³¹I治疗、甲状旁腺功能相关回顾性队列、病例对照、基础机制研究文献, 归纳¹³¹I治疗相关毒副作用、甲状旁腺损伤发生机制、功能评估手段、影响PTH波动的危险因素及防治策略, 对比国内外现有研究争议与局限性。结果: 现有研究对¹³¹I是否影响甲状旁腺功能尚未形成统一结论。术后甲状旁腺功能异常首先与手术相关因素密切相关, 包括甲状旁腺误切、热损伤及供血血管受损; 在此基础上, ¹³¹I治疗可能产生额外的近距离辐射暴露及旁观者效应, 从而使部分患者PTH短暂下降或功能恢复延迟。女性、大剂量¹³¹I及甲状旁腺原位保留数量不足可能与功能异常相关, 但现有研究多为回顾性研究, 且混杂因素控制有限, 因此尚难将PTH变化完全归因于¹³¹I治疗。结论: 术后甲状旁腺功能减退的主要原因仍是手术直接损伤, ¹³¹I更适合作为可能的附加影响因素进行评价, 而不宜将两者混为一谈。现阶段尚缺乏充分证据证明¹³¹I可独立导致甲状旁腺结构性损伤, 因此暂不建议将甲状旁腺功能减退列为¹³¹I治疗禁忌。临床应在治疗前评估甲状旁腺基线功能, 术中规范保留甲状旁腺及其血供, ¹³¹I治疗后进行短期及长期PTH动态监测, 并通过前瞻性研究进一步明确¹³¹I的独立效应。

关键词

分化型甲状腺癌, ¹³¹I, 甲状旁腺, 甲状旁腺激素, 甲状旁腺功能减退

*通讯作者。

Advances in the Study of the Correlation between Postoperative Radioiodine Therapy and Parathyroid Hormone Levels in Differentiated Thyroid Cancer

Shuo Gu¹, Fei Wang², Chunyu Li³, Lili Cui², Junyu Zhao^{2,4*}

¹School of Clinical Medicine (Affiliated Hospital), Jining Medical University, Jining Shandong

²Department of Endocrinology and Metabology, The First Affiliated Hospital of Shandong First Medical University & Shandong Provincial Qianfoshan Hospital, Shandong Key Laboratory of Rheumatic Disease and Translational Medicine, Shandong Institute of Nephrology, Jinan Shandong

³Geriatrics Department of Jinan (Yinfeng) Hospital, Jinan Shandong

⁴Department of Endocrinology and Metabology, Shandong Provincial Qianfoshan Hospital, Cheeloo College of Medicine, Shandong University, Jinan Shandong

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Abstract

Objective: Postoperative radioiodine-131 (¹³¹I) ablation is a standard adjuvant therapy for differentiated thyroid carcinoma (DTC). The anatomical adjacency between parathyroid glands and thyroid tissue may lead to radiation injury of parathyroid glands and abnormal parathyroid hormone (PTH) secretion after ¹³¹I treatment. This review systematically summarizes the effects of postoperative ¹³¹I therapy on parathyroid function and PTH levels, related mechanisms, risk factors and clinical interventions, so as to provide evidence for safe clinical application of radioiodine therapy. **Methods:** Domestic and foreign literatures including retrospective cohorts, case-control studies and basic mechanism researches concerning DTC, ¹³¹I therapy and parathyroid function were retrieved. We sorted out radiation side effects of ¹³¹I, injury mechanism of parathyroid glands, functional evaluation methods, risk factors of PTH fluctuation and prevention strategies, and compared controversies and limitations of existing studies. **Results:** Consistent conclusions have not been reached regarding the effect of ¹³¹I therapy on parathyroid function. Postoperative parathyroid dysfunction is primarily related to surgery, including inadvertent resection, thermal injury, and disruption of the parathyroid blood supply. On this basis, ¹³¹I may exert an additional effect through local radiation exposure and radiation-induced bystander signaling, potentially causing transient PTH decline or delayed functional recovery in some patients. Female sex, higher ¹³¹I dose, and fewer parathyroid glands preserved in situ may be associated with abnormal PTH, but most available studies are retrospective and incompletely control for confounding factors; therefore, the observed PTH changes cannot yet be attributed independently to ¹³¹I therapy. **Conclusion:** The major cause of postoperative hypoparathyroidism remains direct surgical injury, whereas ¹³¹I should be considered a potential additional factor rather than being conflated with surgical injury. Current evidence is insufficient to establish that ¹³¹I independently causes structural parathyroid injury; therefore, hypoparathyroidism should not currently be regarded as a contraindication to ¹³¹I therapy. Baseline parathyroid function should be assessed before treatment, parathyroid glands and their blood supply should be carefully preserved during surgery, and PTH should be monitored dynamically after ¹³¹I therapy. Prospective studies are needed to determine the independent effect of ¹³¹I exposure.

Keywords

Differentiated Thyroid Cancer, ^{131}I , Parathyroid Glands, Parathyroid Hormone, Hypoparathyroidism

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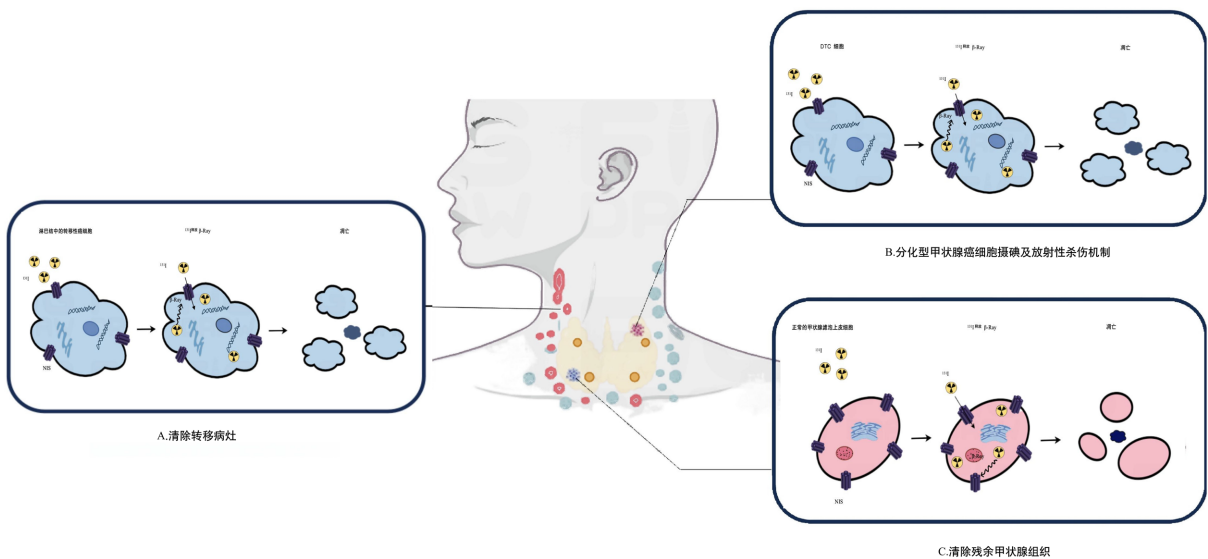
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1. 分化型甲状腺癌患者 ^{131}I 治疗相关毒副反应

^{131}I 治疗的作用基础是正常甲状腺滤泡上皮细胞及 DTC 细胞膜表面表达的钠碘同向转运体(sodium iodide symporter, NIS)，该转运体可在 TSH 刺激下摄取足量 ^{131}I [1]。被摄取的 ^{131}I 可释放高能 β 射线，残余甲状腺组织、微小病灶及转移灶吸收射线后可通过电离效应被破坏，从而实现良好的 ^{131}I 治疗效果[2] (图 1)。但临床实践中已发现多种与放射相关的不良事件，早期不良反应包括恶心呕吐、涎腺炎、骨髓抑制、精子减少、妊娠及卵巢相关并发症、干眼症及鼻泪管阻塞等[3][4]；晚期不良反应包括白血病及第二原发性肿瘤、放射性肺纤维化、慢性干眼症及遗传效应等[5][6]。因此，仍需进一步探索 ^{131}I 治疗可能存在的其他不良反应。



注：NIS：钠碘转运体；RAI：射线；DTC：分化型甲状腺

Figure 1. Schematic diagram of postoperative ^{131}I therapy
图 1. 术后 ^{131}I 治疗示意图

^{131}I 对甲状旁腺功能的不良影响最早于 1983 年被报道[7]，此后也有 ^{131}I 治疗后出现甲状旁腺功能减退并后续恢复的特殊病例报告。由于甲状腺术后残余组织与甲状旁腺解剖位置邻近， ^{131}I 治疗过程中残余组织吸收的 ^{131}I 衰变产生的 β 射线最大射程可达 2.4 mm，理论上可能对甲状旁腺造成电离辐射损伤[8]。近年来陆续有其他关于 ^{131}I 对甲状旁腺功能不良影响的回顾性研究发表，研究结果差异较大。Guyen 和 Glazebrook 研究发现， ^{131}I 治疗后甲状旁腺激素(PTH)水平出现短暂性显著下降，但无低钙血症症状[8][9]。刘刚等也得到了相同的研究结果，同时发现 PTH 水平变化与年龄、残余甲状腺组织量等因素无关，但与

患者性别相关, 女性患者对 ^{131}I 治疗更敏感。但 Zhao 等通过观察性研究指出, ^{131}I 治疗过程中不会损伤甲状旁腺功能, 该研究未纳入年龄、性别、TNM/AJCC 分期、甲状旁腺基础状态、 ^{131}I 剂量等因素作为协变量[10]。Zhang 等也报道 81 例 DTC 患者接受 ^{131}I 治疗 3 个月后, 甲状旁腺功能未受明显影响, 且 PTH 水平变化在不同性别、年龄、T 分期、N 分期患者中无显著差异[11]。袁飞等创新性地对比了同期接受 ^{131}I 治疗组与未接受 ^{131}I 治疗组的甲状旁腺功能相关指标水平, 探索了 ^{131}I 剂量、治疗间隔时间、甲状旁腺自体移植对甲状旁腺功能的影响[12]。随访后发现, 甲状腺乳头状癌术后首次放射性碘治疗短期内对甲状旁腺功能无显著不良影响, 且 ^{131}I 治疗后 3 个月 PTH 水平升高无独立危险因素。同时也有少数病例报告显示, 少量患者 ^{131}I 治疗后出现甲状旁腺腺瘤[13]。

2. ^{131}I 治疗影响甲状旁腺功能的机制

甲状腺及其他甲状腺外组织, 包括胃、肠道、乳腺中碘的主动转运由 NIS 介导[14]-[16]。目前主流观点认为甲状旁腺不具备明显聚碘能力, 甲状旁腺细胞中也未检测到明确的 NIS 表达。因此, ^{131}I 对甲状旁腺的潜在影响更应理解为“邻近残余甲状腺组织摄取 ^{131}I 后产生的附加辐射效应”, 而非甲状旁腺自身摄取 ^{131}I 所致。首先应强调, 甲状腺术后 PTH 下降最常见的基础原因仍是手术直接损伤, 包括甲状旁腺误切、热损伤以及供血血管受损; ^{131}I 治疗是在此基础上可能叠加的因素。残余甲状腺组织摄取 ^{131}I 后, β 射线可在局部短距离内沉积能量, 若甲状旁腺位于其有效射程范围内, 则理论上存在直接近距离辐射暴露的可能[8]。另一方面, 即使甲状旁腺细胞未直接受到明显辐射, 其邻近的受照细胞仍可能通过“辐射诱导旁观者效应”向未直接受照细胞传递应激信号[17]。

从细胞和分子层面看, 旁观者效应并非单一通路, 而是由多种细胞间通讯共同介导。残余甲状腺组织受 ^{131}I β 射线照射后, 可发生氧化应激、DNA 损伤及线粒体功能改变, 并诱导 ROS/RNS、NO 及炎症相关细胞因子等应激信号释放; 这些信号可通过旁分泌途径作用于邻近甲状旁腺细胞。与此同时, 缝隙连接介导的细胞间通讯以及细胞外囊泡/外泌体所携带的蛋白质、脂质和 miRNA 等, 也可能参与损伤信号传播。受体细胞接收到这些信号后, 可进一步激活 NF- κ B、MAPK 等应激和炎症通路, 导致氧化应激增强、DNA 损伤、细胞周期改变、凋亡或衰老, 并可能影响甲状旁腺主细胞的功能状态及 PTH 分泌。上述机制目前主要来自一般放射生物学和体外/动物研究, 尚缺乏直接针对 ^{131}I 治疗后人甲状旁腺组织的实验证据, 因此在本研究领域仍应表述为“可能机制”, 而不能视为已经证实的因果链条[17]。

3. 甲状旁腺功能减退相关临床表现

甲状旁腺是人体的内分泌腺体, 多数为扁平卵圆形小体, 外观为棕黄色米粒状或压扁的黄豆状, 直径约 3~5 mm, 外包薄结缔组织被膜, 常被甲状腺组织、淋巴组织及周围脂肪组织覆盖。甲状旁腺位于甲状腺邻近区域[18], 多数位于甲状腺真假被膜的结缔组织内, 共 4 枚, 左右各 2 枚。但有一定比例人群的甲状旁腺数量并非 4 枚, 数量差异较大, 可少于或多于 4 枚。甲状旁腺细胞或其血供受损会导致甲状旁腺功能减退, 进而引发低钙血症[19]。

陆生脊椎动物的钙稳态由甲状旁腺调控, 甲状旁腺的嗜酸性细胞和主细胞合成并分泌 PTH。PTH 半衰期短, 是评估甲状旁腺功能最可靠的标志物。在人体内, PTH 与降钙素、维生素 D 共同构成血清钙离子调节系统, 维持稳定的血清钙水平对肌肉功能、神经传导、酶及激素分泌等多项生理功能至关重要[20]。甲状旁腺表达钙感受器, 可监测血清钙水平[21]。当受体检测到低钙水平时, 甲状旁腺分泌 PTH, PTH 与成骨细胞上的 G 蛋白偶联受体相互作用, 促使长骨中的钙释放进入血液。在肾脏中, PTH 可增加亨利袢升支粗段及远端小管对钙的重吸收, 同时增加尿无机磷酸盐排泄[22]。因此 PTH 在钙磷代谢稳态、细胞凋亡及骨代谢中发挥重要作用, 可保障内环境相对稳定。当甲状旁腺功能异常时, 钙磷平衡被破坏,

会引发甲状旁腺疾病，如甲状旁腺功能亢进或甲状旁腺功能减退。

甲状旁腺功能减退的临床患病率低，病因相对有限。其中甲状腺术后甲状旁腺功能减退被认为是甲状腺全切术最常见的并发症[23]，一般可分为两类：第一类为持续性甲状旁腺功能损伤及低钙血症，既往文献报道其发生率低于 13%，但多数研究报道发生率不足 1%；第二类为短暂性甲状旁腺功能减退及低钙血症，发生概率约为 30% [24]。意外切除、热损伤及甲状旁腺血供中断通常被认为是甲状腺术后甲状旁腺功能减退的常见原因[25]。

甲状旁腺受损时可导致 PTH 水平下降，轻症患者表现为口周、手足针刺感及麻木，伴情绪激动或易怒；重症患者可出现肢体抽搐、喉支气管及膈肌痉挛、癫痫发作、窒息、心律失常，甚至危及生命。甲状旁腺功能减退的具体临床表现包括：1) 急性低钙血症：典型表现为手足抽搐，有时伴喉痉挛、喘鸣，甚至惊厥或癫痫发作；2) 慢性低钙血症：存在一定程度的神经肌肉兴奋性增高；3) 慢性高磷血症：血管、神经、肾脏等软组织器官异位矿化；4) 肌肉、神经及精神表现：乏力、肢体及口周麻木，神经肌肉兴奋性增高引发肌肉痉挛，如手足抽搐、喉痉挛喘鸣、支气管痉挛等。

因此甲状旁腺功能减退通常会对患者的社会功能、幸福感及生活质量产生显著负面影响。

4. 甲状旁腺功能评估方法

目前检测甲状旁腺功能的主要方法是采集甲状旁腺功能相关指标，包括 PTH、血清钙、镁、磷、25-OH-VD。甲状旁腺激素水平采用免疫荧光法检测为更好地评估甲状旁腺功能，袁飞等在研究中确定了相应指标的参考范围[12]：PTH 参考范围为 1.6~6.9 pmol/L，25-OH-VD、血清钙、血清镁参考范围分别为 47.7~144 nmol/L、2.1~2.7 mmol/L、0.67~1.04 mmol/L，血清磷参考范围为 0.81~1.45 mmol/L。甲状旁腺功能减退的定义为甲状旁腺激素水平低于 1.6 pmol/L。

约 90% 的成年患者甲状腺全切术后即刻会出现甲状旁腺功能减退，术后 4~6 周内恢复，即短暂性甲状旁腺功能减退，其余患者会进展为长期甲状旁腺功能减退，以血清 PTH 水平降低为特征。成人术后甲状旁腺功能减退超过 6 个月通常被诊断为永久性甲状旁腺功能减退，但该诊断标准仍存在争议[26] [27]。

5. ^{131}I 治疗后甲状旁腺效应相关影响因素

甲状旁腺功能减退是甲状腺全切术后常见的并发症，通常 1 个月内可恢复。研究表明，甲状旁腺功能恢复是一个动态过程，术后可能需要较长时间才能完成，有报道显示术后最长 16 年仍可出现甲状旁腺功能恢复[28]。与甲状旁腺功能恢复延迟相关的最核心因素包括原位保留甲状旁腺数量及长期低钙血症患者的甲状旁腺自体移植情况[29] [30]。

Sitges-Serra A 报道，原位保留甲状旁腺数量是延迟性甲状旁腺功能减退患者甲状旁腺功能恢复概率的关键变量[31]，但该研究在评估甲状旁腺功能恢复持续时间风险时未考虑 ^{131}I 治疗的影响。Abuduwalli 等进一步发现， ^{131}I 治疗与术后持续性甲状旁腺功能减退相关[32]，指出 ^{131}I 治疗可显著延长迟发性甲状旁腺功能减退患者的甲状旁腺功能恢复时间，导致功能恢复延迟。由于 ^{131}I 治疗多在术后 1 个月进行，且可多次实施， ^{131}I 对甲状旁腺的持续损伤可能进一步延长患者的甲状旁腺功能恢复进程，导致被诊断为“永久性甲状旁腺功能减退”，但该诊断至少应在术后 12 个月谨慎作出，尤其是接受 ^{131}I 治疗的患者，因为长期替代治疗会给患者带来经济负担和心理影响。

6. 甲状旁腺功能减退相关治疗

目前甲状旁腺功能减退尚无根治手段，患者需每日服用活性维生素 D、钙剂和/或重组 PTH 以调节血钙水平。Luo 等在一项纳入 559 例患者的回顾性研究中发现[33]，甲状旁腺原位保留比例是甲状腺癌术后

短暂性甲状旁腺功能减退、持续性甲状旁腺功能减退及术后低钙血症的独立危险因素，术中甲状旁腺损伤可由血供障碍、热损伤或意外切除导致[23]。因此如何尽可能避免甲状旁腺损伤尤为重要：手术过程中应采用成熟的甲状旁腺识别技术及示踪剂，仔细识别并保留每一枚甲状旁腺及其供血血管；对于无法保留原解剖位置或术中意外切除的甲状旁腺，应采用自体移植术，最大程度避免甲状旁腺功能减退的发生。 ^{131}I 治疗期间及治疗后应定期随访患者血清甲状旁腺激素水平，及时发现并处理甲状旁腺功能减退。此外 Shieh 等指出，游离 25-OH-VD 水平升高与 PTH 水平降低相关[34]，建议临床医生更多关注 25-OH-VD 水平较低的患者，在 ^{131}I 治疗早期给予骨化三醇补充。

7. 总结与展望

尽管 ^{131}I 治疗可使中高危及部分低危 DTC 术后患者获益[35]，但现有证据更支持将其对甲状旁腺的影响理解为潜在的“附加效应”，而非术后甲状旁腺功能减退的首要原因。甲状腺术后 PTH 下降首先应结合手术范围、甲状旁腺原位保留数量、血供情况及术后早期 PTH 变化判断手术直接损伤；对于在此基础上接受 ^{131}I 治疗的患者，则需进一步关注其是否出现额外的 PTH 下降或功能恢复延迟。现有研究仍不足以证明 ^{131}I 对甲状旁腺具有独立的长期损伤效应，因此不能仅依据观察到的术后 PTH 异常将其直接归因于 ^{131}I 治疗，也不能据此将甲状旁腺功能减退列为 ^{131}I 治疗禁忌证。

目前国内外相关研究多为回顾性研究，且患者的手术范围、甲状旁腺保留数量及血供状态、维生素 D 水平、钙剂/活性维生素 D 使用、 ^{131}I 剂量及重复治疗等因素存在明显异质性，因而难以分离手术直接损伤与 ^{131}I 潜在附加影响。未来建议开展前瞻性队列研究：纳入甲状腺手术后均原位保留 2~3 枚且确认具有血供的甲状旁腺、术前及术后早期 PTH 和血钙等指标基本一致的 DTC 患者，根据是否接受 ^{131}I 治疗分为暴露组与对照组，进行长期动态随访。建议在术前、术后 24~48 h、1 周、1 个月、3 个月、6 个月、12 个月及 24 个月检测 PTH、血钙、磷、镁和 25-OH-VD，并在每次 ^{131}I 治疗前后增加时间点采样；同时记录手术范围、中央区淋巴结清扫、甲状旁腺原位保留数量及血供、是否自体移植、 ^{131}I 给药剂量及累计剂量、维生素 D 和钙剂使用等变量。以 PTH 长期变化轨迹、PTH 恢复时间及持续性低 PTH 发生率为主要结局，采用多变量纵向分析或倾向评分等方法控制混杂因素，从而更有可能识别 ^{131}I 对甲状旁腺功能的独立效应。机制研究方面，可进一步结合动物实验和体外细胞模型，检测 ROS/RNS、炎症因子、NF- κB /MAPK 信号、DNA 损伤标志物及细胞外囊泡等指标，以验证旁观者效应在 ^{131}I 相关甲状旁腺功能变化中的作用。

声 明

作者声明本研究不存在任何可能被视为潜在利益冲突的商业或财务关系。

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