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上颌窦气化与口腔医学诊疗的临床关联及研究进展

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摘 要

上颌窦作为人体最大的副鼻窦，其解剖结构与气化特征直接影响口腔医学临床诊疗策略与操作风险。本文通过系统回顾国内外研究，整合上颌窦解剖生理特征、发育动态及气化异常的临床证据，系统归纳种族差异、性别年龄、牙列状态、鼻腔解剖及病理因素等多维度气化调控机制，并深入剖析其与口腔内科、外科、种植及正畸治疗的跨学科关联，为临床精准评估上颌窦相关风险、优化诊疗方案及减少并发症提供了关键理论支撑。

关键词

上颌窦气化，口腔医学，解剖变异

Clinical Associations and Research Progress of Maxillary Sinus Pneumatization in Oral Medicine Diagnosis and Treatment

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Abstract

The maxillary sinus, being the largest paranasal cavity in humans, critically dictates clinical

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decision-making and procedural risk stratification in dental practice through its anatomical complexity and pneumatization patterns. This study synthesizes global evidence to delineate the sinus's morphophysiological properties, developmental trajectory, and clinically significant pneumatization anomalies. A multidimensional regulatory framework is established, incorporating ethnic variations, sex- and age-related modifications, dentoalveolar architecture, nasal cavity configurations, and disease-mediated alterations. Through rigorous interdisciplinary analysis, the investigation elucidates the sinus's pathophysiological interplay with endodontic procedures, maxillofacial surgery, implant placement protocols, and orthodontic biomechanics. These insights advance evidence-based protocols for precision diagnostics, risk mitigation strategies, and complication management in maxillary sinus-associated interventions, thereby establishing a foundational paradigm for optimized therapeutic outcomes in contemporary stomatological practice.

Keywords

Maxillary Sinus Pneumatization, Oral Medicine, Anatomical Variation

1. 引言

上颌窦又称“海莫尔窦”，最早是在1651年由纳撒尼尔•海莫尔(Nathaniel Highmore)提出的[1]。上颌窦左右各一，是4组副鼻窦中气化体积最大者[2]。出生时上颌窦为一个大部分充满液体的裂隙状结构，随着个体的生长发育，上颌窦窦腔内的液体吸收并替换为气体，在成骨、破骨程序的相互作用下窦腔不断扩大，这个过程称之为上颌窦气化[3]。上颌窦的底壁由上颌骨牙槽突和部分硬腭组成，与口腔最为密切。底壁的前后范围通常覆盖上颌前磨牙和磨牙根尖区域，而上颌第一磨牙和第二磨牙是根尖与上颌窦关系最为密切的牙位之一，甚至出现根尖进入上颌窦内的情况[4] [5]，这会影响口腔各个学科的诊疗决策及过程。本文将整合上颌窦解剖生理特征、发育过程及异常气化的现象，深入剖析其与口腔多学科之间的关联，并系统归纳其影响因素，为临床诊疗提供理论依据。

2. 上颌窦的解剖生理

上颌窦位于上颌骨体内，呈锥体形，两边各一，是副鼻窦中体积最大的一组。成人上颌窦的平均容积约为15 mL，平均体积约36.4 cm^2，长度约38~45 mm，高度约36~45 mm，宽度约25~35 mm [6] [7]。其形态随年龄增长而变化，出生时仅为裂隙状，青春期快速发育，20岁左右定型[8]。上颌窦的形态变异较大，可能表现为单房或多房结构。研究表明，约13%至35.3%的上颌窦有分隔的存在(septa)。它们可以位于上颌窦的任何区域，其平均长度在2.5到12.7 mm之间，这些分隔可能影响手术入路及治疗效果[9] [10]。

上颌窦由多个骨性壁构成，各壁及其毗邻结构对临床操作具有重要意义[1]。前壁由上颌骨前面构成，中部尖牙窝处凹陷而骨质较薄，眶下孔穿行眶下神经血管束。后外侧壁毗邻颞下窝及翼腭窝，内含上牙槽后血管神经。该壁与颧弓及咀嚼肌相邻，感染可能向深部扩散。内侧壁构成鼻腔外侧壁下部，后上方有自然开口通中鼻道，位于半月裂孔后部。约30%人群存在副开口，这可能影响鼻窦引流及感染扩散[11]。上壁分隔窦腔与眼眶，上壁的骨质薄弱区域是眼眶底骨折的常见部位，外伤易致眶内容物疝入窦内。此外，上壁与眶下神经管相邻，手术中需避免损伤眶下神经。底壁由上颌骨牙槽突构成，第二前磨牙至第

三磨牙根尖与上颌窦底毗邻甚至伸入上颌窦内，因此上颌窦底壁与口腔医学领域的关系尤为密切。

上颌窦的动脉血供主要来自上颌动脉的分支，包括蝶腭动脉、眶下动脉、上牙槽后动脉及腭大动脉等。这些动脉在黏膜下层形成丰富的血管网，为窦腔提供营养。此外，上颌窦的动脉与鼻腔及牙槽区的血管存在广泛吻合，这可能增加相关区域手术时的出血风险。上颌窦的静脉回流至面静脉，后经上颌静脉汇入颈内静脉。上颌窦的神经支配主要来自三叉神经上颌支发出的上牙槽神经(前、中、后支)及眶下神经，这些神经分支穿行于窦壁骨结构，支配窦粘膜[1]。

3. 上颌窦气化模式及异常气化

3.1. 上颌窦的发育

人类副鼻窦的发育始于鼻腔向对应骨(额骨、上颌骨、筛骨、蝶骨)内的凹陷。最早开始发育的是上颌窦，在胚胎发育第 17 天即可观察到其雏形。副鼻窦的发育进程自妊娠第 3 周启动，并持续至成年早期。胚胎第 4 周时，外胚层细胞增殖并向中线迁移形成脊索。该脊索最初位于胚胎盘尾端区域，随后旋转至原始前肠后方。与此同时，第一咽弓的背侧部分形成上颌突，向前延伸至发育中的眼球下方，最终构成上颌骨[12]。

至胚胎第 2 个月末，上颌窦开始以下鼻甲上方的凹陷形式出现，并向侧方生长。第 12 周时，黏膜从中鼻道外侧壁外凸，逐渐使鼻上皮侵入全部上颌间充质[13]。新生儿时期，上颌窦表现为狭长的裂隙状结构，内部主要含液体，位于上颌骨内侧壁，其最大径线位于矢状面且小于 8 毫米。通过常规影像学检查通常无法显示新生儿窦腔，此时原始含气窦腔容积约为 6~8 cm^3，矢状面上前后径最大[8]。

出生后 1 年末，上颌窦外侧缘延伸至眶内侧壁下方；4 岁时，其外侧边界扩展至眶下窝；至 9 岁时，窦腔向下抵达硬腭平面，外侧达上颌骨体[14]。尽管上颌窦不同发育阶段的时序存在显著个体差异，但这些阶段在时间上通常紧密关联[15]。上颌窦持续向下扩张，伴随上颌牙槽骨气化过程，至 12 岁时窦底降至鼻腔底水平，最终窦底低于鼻底约 4~5 毫米[16]。

3.2. 上颌窦发育不良

上颌窦发育不良(Maxillary sinus hypoplasia, MSH)是一种少见的解剖异常，文献报道其发生率约为 1%~11% [17]，解剖学表现为从轻度的窦腔发育不良到重度的裂隙状窦腔伴勾突结构异常不等[18]。根据 Sirikçi 的定义，上颌窦内外径或前后径的最大值小于眼眶直径的 50%就可以定义为上颌窦发育不良[19]。该病变可源于胚胎期发育障碍，也可继发于创伤性、医源性或结构性因素。尽管主流观点认为漏斗部阻塞导致负压可能引起上颌窦发育不良，但临床发现部分病例在窦口鼻道复合体完全正常的背景下仍存在窦腔发育不全，因此仍有部分学者反对这一观点[20]。大多数 MSH 的患者不会有任何症状，通常是在偶然摄片后才被诊断。但 MSH 可增加鼻内镜手术中眶内壁筛骨纸板骨折(Lamina papyracea injuries)及眼眶穿透风险，因此术前需准确识别此类解剖变异。

3.3. 上颌窦过度气化

若上颌窦内外径或前后径的最大径达到对应眼眶尺寸的 90%及以上，则被定义为上颌窦过度气化(Extensive maxillary sinus pneumatization, EMSP) [21]。上颌窦气化过程可向邻近骨性结构异常延伸，形成以下解剖扩展：向内侧延伸至硬腭，向下方延伸至牙槽突，外侧扩展至颧骨，后方扩展至筛骨[22]。EMSP 无特异性临床表现，其症状谱可从无症状、典型鼻窦炎症状到非典型临床表现。非典型症状可表现为神经性症状、牙源性疼痛、颞下颌关节综合征、屈光异常或非典型面痛，这或许与上颌窦气化范围突破常规解剖界限密切相关[21]。目前对于上颌窦过度气化程度的分型尚未形成共识，Elsayed [23]根据上颌窦底

与上颌后牙根尖的距离，将上颌窦气化程度分为 III 型：I 型为正常气化或气化不良，上颌窦底与上颌后牙根尖距离大于 3 mm；II 型为显著气化，上颌窦底与上颌后牙根尖距离在 0~3 mm 范围内；III 型为过度气化，上颌窦底高度低于上颌后牙根尖。刘锦峰等[24]依据上颌窦底与硬腭的位置关系，将上颌窦底部不低于硬腭的口腔面为 I 型；上颌窦底部低于硬腭且底部完整，牙根未突入上颌窦底部时为 II 型(显著气化)；上颌窦底部低于硬腭且牙根突入到上颌窦底部时为 III 型。

4. 上颌窦气化的影响因素

4.1. 种族差异

种族差异显著影响上颌窦的气化程度，这种差异可能与遗传背景、气候适应性及颅面形态的演化特征相关。在一项对欧洲人和祖鲁人颅骨的研究中[25]，欧洲男性上颌窦比祖鲁男性上颌窦大 62.7%。欧洲女性上颌窦比祖鲁女性上颌窦大 33.8%，说明不同种族的上颌窦容积存在差异。Scott D 等[26]的研究表明撒哈拉以南的非洲人表现出较窄的上颌窦，而极地地区的人则倾向于表现出较宽的上颌窦，欧洲人则介于两者之间。Shea T 等[27]在比较爱斯基摩人、蒙古人、高加索人的上颌窦容积及面部特征时提出，除了大众所广为接受的气候因素外，饮食习惯带来的咀嚼力差异也可能在其中发挥了作用，这些因素共同造就了上颌窦气化程度在种族之间的差异。

4.2. 性别、年龄与左右侧

性别与年龄是影响上颌窦气化进程的重要生理变量。影像学研究表明，男性上颌窦容积普遍大于女性[25] [28]-[34]，例如 Luz J 基于 CBCT 的上颌窦三维重建研究发现，男性平均容积为 19 cm^3，而女性为 15.5 cm^3 [6]。从年龄维度来看，上颌窦气化贯穿整个生命周期，但不同阶段的速率与模式存在显著差异。儿童期(0~12 岁)的窦腔发育以垂直扩展为主，出生时仅为裂隙状，至青春期(12~18 岁)因上颌骨快速生长而显著扩大。成年后(20 岁以上)气化速度减缓直至体积达到最大值。随着年龄的增大，在 20~30 岁之后体积又逐渐减小[13] [29] [35] [36]。此外，现有的研究普遍认为左右侧的上颌窦体积无显著性差异[13] [25] [28] [37] [38]。

4.3. 牙列状态

牙列状态是驱动上颌窦代偿性气化的关键因素，尤其在后牙区表现显著。Julius Wolff [39]于 1892 年提出的 Wolff 定律指出，骨骼的形态与结构会随着力学刺激的变化而动态调整，缺乏力学刺激的区域发生骨吸收，导致骨量减少。Sharan 等[40]的研究发现，缺失牙的一侧上颌窦高度较对侧低 2.18 ± 2.89 mm，而拔牙后同侧上颌窦高度会降低 1.83 ± 2.46 mm。其他许多研究也支持这一观点[41] [42]。牙齿的缺失导致咀嚼功能减退，通过牙槽骨传递至上颌窦底部的机械负荷降低，破骨细胞活跃，上颌窦发生继发性气化[43]。

尽管也有一些研究的结果并不支持这一观点，Wagner F 等[44]使用主成分分析(PCA)研究了 400 名患者的 CT 影像资料，发现与牙列存留的患者相比，牙列缺失的患者虽然牙槽嵴出现了明显吸收，但上颌窦高度没有显著性差异。Hameed S 等[45]对比了 23 例患者拔牙前后的牙槽嵴高度及上颌窦底位置，发现牙槽嵴高度降低了 3.07 ± 2.53 mm，而上颌窦底的高度仅下降了 0.47 ± 0.32 mm，与其他文献报道的降低幅度有所差距。这或许与不同研究间的样本量及测量方法不同有关。

4.4. 鼻腔结构和异常解剖

上颌窦和鼻结构彼此有密切的解剖关系，鼻腔的外侧壁形成上颌窦的内侧壁。多项研究为上颌窦对

鼻腔形态变化的适应性调节提供了初步证据，这些研究普遍表明，鼻腔宽度与上颌窦体积之间存在负相关关系，即狭窄的鼻腔通常伴随较大的上颌窦，而较宽的鼻腔则伴随较小的上颌窦[26] [27] [46] [47]。

在鼻窦区域可以看到相当复杂的结构和许多解剖学变化，包括鼻中隔偏曲、泡状鼻甲、鼻中隔棘突、钩突气化、中鼻甲肥大、哈勒气房等。这些异常解剖会改变鼻腔内气流流量及形式，进而影响上颌窦的通气状态和气体交换[48]，这可能使上颌窦气化程度出现变异[49]。Kalabalik 和 Orhan 的研究表明，鼻中隔偏曲同侧的上颌窦体积小于对侧[50] [51]。根据鼻中隔偏曲的严重程度进行分组后，不同亚组之间的上颌窦体积之间也存在差异[50] [52]。然而，许多研究者则得出了相反的结论，认为鼻中隔偏曲对上颌窦体积无明显影响[30] [53] [54]。同样的矛盾结论也发生在了泡状鼻甲上，Al-Rawi 的研究指出有泡状鼻甲的病例上颌窦体积显著大于正常组，且双侧泡状鼻甲的组别大于单侧组[53]。然而更多的研究者并没有发现泡状鼻甲和上颌窦体积之间的关系[32] [48] [49]。以上鼻腔异常解剖与上颌窦气化程度关联性研究的结论分歧可能与研究方法的异质性有关，鼻中隔偏曲分组标准的主观性和上颌窦体积测量方法的不同都会影响结论的得出。

4.5. 病理状态

占位性病变与慢性炎症是导致病理性气化的主要因素。上颌窦的可塑性体现在其能够通过扩张和压缩窦壁来适应内部或外部缓慢生长的占位性病变，上颌窦粘液囊肿、牙源性囊肿等非侵袭性良性病变通过挤压和压迫上颌窦壁引起其气化改变；侵袭性浸润性病变如成釉细胞瘤、恶性肿瘤等可引起骨破坏，其影像学表现常同时具有骨质重塑和侵蚀的混合特征；而累及上颌骨髓腔间隙的病变(如纤维骨性病变、遗传性贫血及骨发育不良)会逐渐导致上颌窦腔闭塞[12]。Sonone 的一项前瞻性横断面研究发现，慢性鼻窦炎的患者上颌窦各个骨壁都会出现增厚，从而导致上颌窦体积减少[55]，而这与 Kim 等[56]和 Cho 等[57]的研究结果一致。

5. 上颌窦气化与口腔医学的联系

上颌窦底部通过牙槽突与牙列相连，通常其间仅有一层菲薄的骨质。根据文献报道，上颌第一磨牙与第二磨牙的牙根与上颌窦之间的距离最近[58] [59]。与当上颌窦出现过度气化时，牙根与上颌窦之间的距离进一步降低，甚至出现牙根伸入上颌窦内的情况。因此上颌窦的气化程度会影响口腔医学各学科诊疗时的决策。

5.1. 口腔内科领域

根尖周病变在累及甚至破坏上颌窦底壁时，容易刺激施奈德膜使其出现增厚，甚至发生上颌窦炎[60]，大约 10%至 12%的上颌窦炎病例是由牙源性感染引起的[61]。根尖周病变中的细菌及毒素可直接通过丰富的血管吻合、多孔的骨髓腔或淋巴系统渗透至上颌窦，从而感染窦黏膜[62]。当根尖周病变越严重时，细菌与毒素数量的增加会导致根尖周炎严重程度加重，进而提高上颌窦病变的可能性[63]。甚至有文献报道，牙源性感染可通过上颌窦快速扩散，进而引发眶周蜂窝织炎、视力丧失甚至危及生命的海绵窦血栓[64] [65]。

对牙根靠近上颌窦的牙齿进行根管治疗时，上颌窦出现并发症的可能也会较牙根远离上颌窦者高出许多。虽然根管治疗要求操作过程中使用的器械及材料都应当局限在根管之内，但实际操作过程中医生总是不可避免地会有概率将根管预备产生的碎屑、根管冲洗/根管消毒的药物、根管充填的材料等超出根尖，进入根尖周组织引起炎症间接影响上颌窦，甚至直接进入上颌窦内引起严重的并发症[62] [66] [67]。过度气化的患者在接受上颌后牙区的根尖手术时，也更加容易出现上颌窦穿孔的情况[68]。

而与之相对的，上颌发育不良的患者由于后牙区牙槽骨高度充足，在进行根管治疗和根尖手术治疗的并发症更少[69]。

5.2. 口腔外科与种植领域

口腔上颌窦交通(OSC)指口腔与上颌窦之间的软硬组织缺失形成病理性通道，是上颌后牙区拔牙后的常见并发症之一，且根尖距离上颌窦越近时发生 OSC 的概率越高[70]。如果口腔上颌窦交通长时间存在，口腔微生物易侵入上颌窦，可能导致窦膜慢性炎症，甚至形成永久性上皮化的口腔窦瘘，显著增加鼻窦炎风险[71]。当上颌窦过度气化时，根尖与上颌窦黏膜的关系十分密切甚至直接接触，此时医生绝对不能忽视拔牙后 OSC 的风险。

对于口腔种植领域，上颌后牙区的种植手术常常需要面对剩余骨高度不足的情况，其中拔牙后上颌窦的继发性气化和牙槽骨的改建共同发挥了作用[44]。研究表明，剩余骨高度会显著影响种植治疗随时间推移的成功率[72]。此时，为了让骨高度可以安放植体，上颌窦底提升术(Maxillary sinus floor augmentation)往往是种植手术中必要的步骤，其经典方法包括经上颌窦外侧壁开窗的外侧入路提升术和经牙槽嵴顶入路提升术[73]。但与简单种植相比，更加复杂的操作既提高了对医生的技术要求，又增加了黏膜穿孔、出血、感染等并发症的风险[74]。

5.3. 口腔正畸领域

当牙根与上颌窦关系密切时，正畸治疗也会面临更多的挑战。Wehrbein 等[75]的研究显示，与上颌窦底更为水平的组别相比，如果上颌窦底的气化部分延伸至要移动的牙根前方时，牙根会出现明显更高程度的倾斜(约 10 度)，且倾斜程度与上颌窦的气化程度存在统计学上的相关性。甚至在 Oh 等的病例中牙根前治疗的前 3.5 年间出现了绝对的皮质骨锚定[76]。此外，众多研究报道了当在上颌窦骨皮质内移动牙根时，会出现明显的牙根外吸收[77]。为了尽量降低发生以上情况的风险，使用恒定的轻到中等的正畸力以及更慢的移动速度是目前更为推荐的治疗策略[78]。

6. 总结与展望

现有的研究对于上颌窦的生理解剖、发育过程、病理变化都等已经有了较为完善的研究，但对于影响上颌窦气化的后天因素尚还存在许多争论，机制也未完全阐明。未来研究需进一步深入探索异常气化的机制研究，寻找精准有效的干预手段，以期为口腔临床实践提供更具指导价值的科学依据。

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