

Review Article

New Insight on Pathophysiology, Diagnosis, and Treatment of Odontogenic Maxillary Sinusitis

Jianhua Zhu,¹ Wei Lin,² Wenwen Yuan,³ and Lili Chen⁴ 

¹Graduate School of Zhejiang University, China

²Department of Otorhinolaryngology, Litongde Hospital, China

³Department of Stomatology, Litongde Hospital, China

⁴Department of Stomatology, The Second Affiliated Hospital of Zhejiang University, Zhejiang, China

Correspondence should be addressed to Lili Chen; y216180119@zju.edu.cn

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Odontogenic maxillary sinusitis is often ignored by otolaryngologists, dentists, and imageological diagnosis doctors. Traditional treatments are often frustrating for refractory maxillary sinusitis and odontogenic maxillary sinusitis. In the last few years, new progress has been made in the diagnosis, pathophysiology, and treatment of odontogenic maxillary sinusitis. Periodontitis, polyposis, and iatrogenesis are regarded as causes of odontogenic maxillary sinusitis. Dental implant dislocation into the maxillary sinus and augmentation are the main cause of iatrogenesis compared to root canal full material. The symptoms are too similar to distinguish odontogenic maxillary sinusitis from chronic rhinosinusitis. Computed tomography is the gold standard for diagnosis, while it is difficult to rule out odontogenic maxillary sinusitis by conventional panoramic radiographs. Cone-beam computed tomography (CBCT) is currently the most broadly used to make diagnosis and differential diagnosis for maxillary sinus disease. The imaging diagnosis, clinical diagnosis, and pathological diagnosis are often not completely in accordance. Most researchers believe that odontogenic maxillary sinus infection results from the spread of apical pathogenic microbial infections: either through the local vascular system, lymphatic system, or the Haval system of the alveolar bone itself. DNA and RNA sequencing of mucosal tissues of maxillary sinus disease confirmed that the mechanisms of odontogenic maxillary sinusitis and nonodontogenic maxillary sinusitis are different. Microbial RNA sequencing of the maxillary sinus also verifies this conclusion. Clinical serum testing of chemical factors has not been widely exploited. It is unknown whether the thickening of the maxillary sinus membrane is the result of pathogen infection or inflammatory mediators. Many doctors have recommended a consensus on multidisciplinary cooperation management, but a global consensus has not yet been reached. Diagnosis methods for odontogenic maxillary sinus disease are diversified. Tissue DNA and RNA sequencing, chemical factor determination, and microbiological DNA and RNA sequencing have appeared, and the misdiagnosis rate has gradually decreased. The pathophysiology of odontogenic maxillary sinusitis and chronic rhinosinusitis is different. The molecular mechanism of the thickening of the Schneider membrane in odontogenic sinus is unknown.

1. Background

The first professor to propose the concept of odontogenic maxillary sinus was William H. Bauer in 1943 [1]. Over the past decades, the concept has evolved, and odontogenic maxillary sinus has become a well-recognized condition in both the dental and otolaryngology communities [2]. Periodontitis, apical periodontitis, and root canal therapy superfilling materials such as silver amalgam and dental tissue are the

main motivation of odontogenic maxillary sinus infection [3, 4]. Since the development of dental implant surgery, many documents have pointed out that iatrogenic factors such as implants and maxillary sinus lifting materials have been accounting for more and more causation [5–9]. Odontogenic maxillary sinusitis has not been documented in the ear, nose, and throat (ENT) practice guidelines in 2012, 2017, and earlier. The “European Position Paper on Rhinosinusitis and Nasal Polyps 2020” mentions the diagnosis of

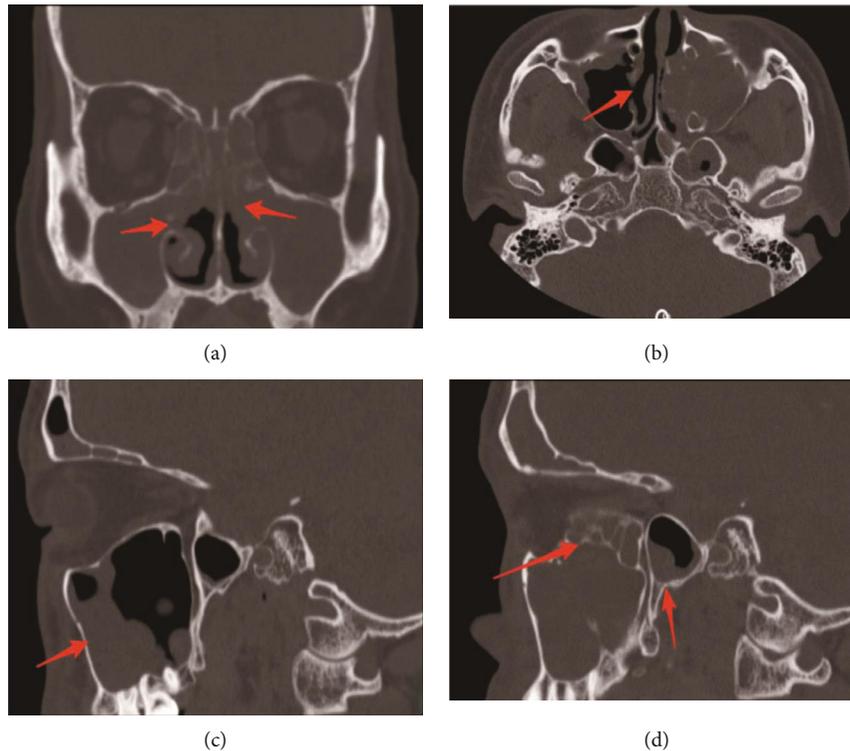


FIGURE 1: Individual with chronic maxillary sinusitis. (a) Bilateral chronic maxillary sinusitis. The arrows show that the lesions scatter over the adjacent tissues finally to the other side of the sinus. (b) The transverse image of the infected area. The arrow shows the infected area. (c) The cross-sectional view of the maxillary sinus on the right. The arrow shows that the lesion spreads to other parts of the maxillary sinuses. (d) The sagittal position of the left maxillary sinus. The arrows show that the lesion spreads to adjacent tissues and finally spreads to other maxillary sinuses.

odontogenic maxillary sinusitis in the classification of rhinosinusitis [10, 11]. Due to the different population bases of various literature statistics, the proportion of odontogenic maxillary sinusitis diagnosis is inconsistent, about 10%-40% [12–14].

2. Diagnosis

It is inaccurate to diagnose with maxillary sinusitis depending on panoramic film alone. Computed tomography (CT) and cone-beam computed tomography (CBCT) are regarded as better options for diagnosing maxillary sinus disease which have been proven in our daily practice [15, 16]. CBCT is currently the most commonly used method to diagnose maxillary sinusitis. However, it is not enough to diagnose maxillary sinusitis according to imaging data alone [16]. It is reported that the pathological examination results are not always in accordance with the CBCT presentation before operation [16–18]. Early differential diagnosis of maxillary sinusitis, sinus mucosa thickening, sinus cyst, maxillary sinus endometrial papillary inverted fold, and sinus mucosal bleeding is not easy [19, 20]. Emphasis with the identification between imaging diagnosis, clinical diagnosis, or pathological diagnosis is important [16]. Dobros and Zarzecka tried to establish a diagnostic model of odontogenic maxillary sinusitis based on symptoms [4].

With the development of science, new diagnostic methods have emerged. More and more literatures which emphasize diagnosis and differential diagnosis utilizing maxillary-related cytokines and chemical factors have gradually increased [21]. Interferon- α ; interferon- γ ; proinflammatory cytokines including brain-derived neurotrophic factor (BDNF), tumor necrosis factor- α (TNF- α), and IL-3; viral-associated chemokines including IP-10 (CXCL10), I-TAC (CXCL11), MCP-3 (CCL7), and MIG (CXCL9); microbial peptides, Toll-like receptors; picric acid receptors; neopterin levels; α -defensins; and prostaglandin D2 all have been selected for laboratory detection for chronic maxillary sinusitis. However, whether it can be used for the differential diagnosis of odontogenic maxillary sinusitis from maxillary sinus needs further study [22–28], whereas cytokines and chemokines are currently detected in local tissues, and detection in serum is relatively rare. Rapoport et al. tried to compare microbial DNA probes with a conventional microbial culture to find the pathogenic microbial as well as chemosensitivity and found that DNA probes are more accurate and reliable than traditional methods [29]. However, the detection efficiency is limited and needs further expansion.

3. Pathophysiology

The pathological characteristics of odontogenic maxillary sinusitis and chronic sinusitis are different [18, 30]. It is

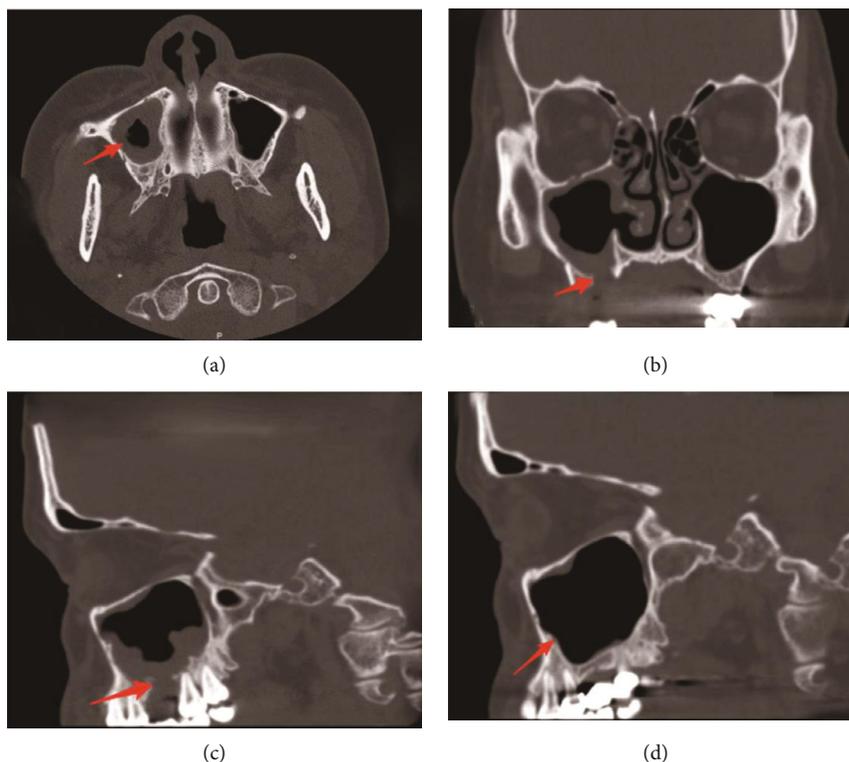


FIGURE 2: Individual with right maxillary sinusitis. (a) The transverse image of the infected area. The arrow shows that the lesions lie in the right maxillary sinus. The left sinus is normal. (b) Comparison sagittal image of left maxillary sinus in the same patient. The arrow shows communication between the oral and the maxillary sinus. (c) The coronal image of the infected area. The patient suffered from odontogenic maxillary sinusitis and had been tooth extracted through the traditional Caldwell-Luc approach. After recurrence, FESS surgery and local mucosal flap transposition were performed transorally. (d) Comparison sagittal image of right maxillary sinus in the same patient.

viewed that the pathophysiology of rhinogenic sinusitis has various reasons leading to the inflammation of the sinus ostium complex, which spreads to the tissues around the ostium, and finally to other maxillary sinus locations, blocking the ability of maxillary sinus mucosal clearance. The cascade of inflammation will lead to damage by the infiltrating cells, causing edema, fluid extravasation, mucus production, engorgement, and sinus obstruction in the process (such as Figures 1(a)–1(d)). The origin of odontogenic maxillary sinusitis is the pathogenic teeth; from the teeth, it diffuses to the maxillary sinus floor, then from the maxillary sinus floor, it spreads to the maxillary sinus ostium and finally to the tissues around the maxillary sinus ostium and other accessory sinuses. The microorganisms even scatter over the orbital and brain [20, 28, 31–35]. The odontogenic maxillary sinusitis can be used as an important basis for distinguishing odontogenic maxillary sinusitis from nonodontogenic maxillary sinusitis (such as Figures 2(a)–2(d)).

In terms of anatomy, it is believed that the veins, arterial connections, and lymphatic system are the main way which make lesion bacteria diffuse from the periodontal to the maxillary sinus. The Haval system in the alveolar bone also acts as an important approach for evaluating the spread of infection [36]. Studies have verified this theory through autopsy, angiography, and radiographic scintigraphy [37, 38]. There are

various reasons for the thickening of the maxillary sinus mucosa. Some authors use CBCT analysis to infer that the thickening of the maxillary sinus mucosa is closely related to periodontal conditions and root apex conditions. The Schneider membranes of many patients have gradually recovered after root canal treatment [39, 40]. It is unclear whether the thickening of the maxillary sinus mucosa and early maxillary sinus inflammation is the result of inflammatory mediators or the scatter of microorganisms to the maxillary sinus [39, 40]. Further research and confirmation in the future are essential.

Maxillary sinusitis is the dysfunction of the host mucosal epithelium under environmental pressure to initiate an inflammatory response. The inflammatory response cascade amplifies the process along corresponding inflammatory mediators. But the complete molecular mechanism is currently unclear [35, 41]. Interestingly, inflammatory mediators are different in various countries, even in different regions of the same country because of different susceptible microorganisms [22, 42]. The inflammatory mediators are different between odontogenic maxillary sinusitis and nonodontogenic maxillary sinusitis. The pathways involved in inflammation are also various [22, 36, 38, 43–45]. In terms of the microorganism, the identification of microbial DNA or RNA sequencing, the detection of virulence genes, and the determination of drug resistance genes are

supplementary methods for routine culture negative results in clinical work. Microbiological gene sequencing showed that microbial communities were constant compared to normal maxillary sinus and chronic maxillary, but the species changed [46, 47]. The microbial dominant bacteria between the odontogenic maxillary sinusitis and the chronic maxillary sinusitis are different by the microbial gene sequencing. For chronic sinus disease, anaerobic bacteria, streptococcus, *Pseudomonas aeruginosa*, or predominate fungi are dominated. Refractory maxillary results from microbial membrane colonization, which has yet to be confirmed by further experiments [48]. The research on proteomics and metabolomics is mainly focused on chronic maxillary, while there are limited data. Limited literature on the differential diagnosis of odontogenic maxillary has been documented [23, 45]. Microbial DNA or RNA detection, proteomics testing, and metabolomics identification are the current and future development directions for the diagnosis and differential diagnosis of microbial infections.

4. Treatment

Elimination of infection source in odontogenic maxillary sinusitis is important for preventing symptom persistence. Antibiotics play a part in treating odontogenic maxillary sinusitis whenever combined with other appropriate treatments. Clindamycin and clavulanate potassium can overcome drug resistance during the treatment of chronic and odontogenic maxillary sinusitis. When maxillary sinus inflammation scatters to adjacent organs, the length of antibiotics is reported for 3 weeks to 3 months, or even longer [49]. Previous evidence showed that 50% of all odontogenic sinusitis pathogens were resistant to clindamycin, making this a poor choice for antibiotic therapy [50]. Craig et al. proposed functional endoscopic sinus surgery (FESS) as the first choice for maxillary sinus treatment prior to endodontic treatment [51]. Tsuzuki et al. proposed that extraction is the prior choice instead of FESS and dental implant [52]. Yoo et al. believe that dental endodontic treatment and medicine are given priority, and FESS is required according to the treatment results [53]. Akiyama et al. proposed that the apical surgery was performed simultaneously with FESS to achieve better results [54]. Aukstakalnis et al. provide grading odontogenic maxillary sinusitis, which is of positive significance for the selection of treatment options and cooperation among multiple departments [55]. Endodontic history and the lesions of odontogenic maxillary sinusitis are candidates for FESS [56]. Craig et al. completed a survey of experts related to odontogenic maxillary sinusitis governance in the United States and achieved consensus on the management of sinus disease. Among them, the participating dentists are relatively scarce, and the numbers of experts participating in the survey are also relatively few [57]. Some researchers proposed that dental surgery should be performed first to eliminate the source of infection, followed by sinus surgery to relieve symptoms and prevent recurrence of sinusitis [56]. The consensus needs further supplementation and improvement.

5. Conclusion

Odontogenic maxillary sinusitis, as a category of chronic maxillary sinus disease, is receiving more and more attention from otolaryngology and maxillofacial surgeons. Dentists and radiologists are gradually joining the management. With gradual comprehension of pathophysiology and microbiology of odontogenic maxillary sinusitis and the improvement of clinical treatment effects, more and more doctors from different countries have accepted the consensus on the management of the disease. Nevertheless, a global management consensus has not yet been signed in, and subsequent studies based on clinical evidence are warrant.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

All authors declare that they have no conflict of interest.

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