International Journal of Medical Sciences And Clinical Research (ISSN – 2771-2265)
VOLUME 04 ISSUE 10 PAGES: 48-55
OCLC – 1121105677
Crossref doi Google Scholar WorldCat MENDELEY
Publisher: Oscar Publishing Services

OSCAR PUBLISHING SERVICES

Journal Website: https://theusajournals.com/index.php/ijmscr

Research Article

ASPECTS OF ETIOLOGICAL FACTORS OF CYST MAXILLO-FACIAL SINUSES

Submission Date: October 02, 2024, Accepted Date: October 07, 2024,
Published Date: October 12, 2024
Crossref doi: https://doi.org/10.37547/ijmscr/Volume04Issue10-09

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ABSTRACT

Upper jaw sinus cysts are a very common disease, often an accidental clinical find upon radiography and computed tomography of the paranasal sinuses. At the same time, there are few reports in the literature about the location of cysts in the frontal and ponal cavities. It is especially difficult to diagnose the cysts of the wedge-shaped cavity, as known radiographic arrangements do not allow them to be diagnosed. The problem of diagnosing paranasal sinus cysts was solved by implementing computed tomography into practice.

KEYWORDS

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Sinusitis, upper humerus, cyst.

INTRODUCTION

Sinuses of odontogenic origin occupy a significant place among inflammatory diseases of the maxillofacial region, and patients with odontogenic maxillary sinusitis make up about 25.8% among patients with inflammatory processes of the maxillary sinus and about 7.6% of patients in maxillofacial hospitals, and among dental and otolaryngological institutions - from 3 to 10% [2].

The disease is a serious general medical and economic problem, as the majority of patients are young and middle-aged people, and in recent years, there has been an increase in the number of patients with chronic obstructive pulmonary disease, ranging from 41% to 77% of all inflammatory processes in the maxillary sinus [12, 17].

Depending on the etiology of the disease, rhinogenic, odontogenic, traumatic and allergic sinusitis of the upper jaw are distinguished. On average, one-third of all maxillary sinusitis is represented by its odontogenic forms [23, 34], which are mainly the result of prolonged existence of a chronic peri-apical infection focus in the area of small and large maxillary teeth (MZ) [4, 11] or errors of doctors in the process of treatment and removal of these teeth [3, 15, 17, 30].All of the above is a pressing dental problem.

The purpose of this study is to analyze the current state, epidemiology, etiology, and pathogenesis of odotogenic maxillary sinusitis.

Despite the improvement of therapeutic and diagnostic equipment, the frequency of CHF among other inflammatory diseases of the maxillofacial region increased from 4-4.2% in the 1970s to 7.6% in the 1990s, and there is a steady trend towards an increase in the number of patients with CHF in recent years [13, 21].A statistical analysis conducted by F.I. Shulman (2003) showed that over the period from 1997 to 2001, the frequency of severe acute respiratory distress syndrome (AVSR) developed as a result of filling material penetration into the upper jaw cavity after endodontic treatment of teeth increased tenfold, indicating a significant impact of the iatrogenic factor on the development and progression of this pathology.

According to literature data [5,9,35] 23.6 to 77.2% of FVV are its perforated forms.

Depending on the etiology of the disease, rhinogenic, odontogenic, traumatic and allergic sinusitis of the upper jaw are distinguished. This division is conditional, as rhinogenic upper jaw sinusitis, developed against the background of acute respiratory disease, can lead to exacerbation of the periapical focus of chronic

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odontogenic infection and secondary infection of the upper jaw sinus mucosa [36]. Therefore, 62% of patients with rhinogenic maxillary sinusitis exhibited odontogenic infection foci [5, 24].

Emergency cases, like rhinogenic ones, are primarily caused by a purulent infection. The leading infectious agents are hemolytic streptococci, pneumococci, staphylococci, as well as opportunistic intestinal bacteria.

Several microbial agents, often a mixed flora: aerobic flora: Staphylococcus anreus, Streptococcus Species, Hemophylus influenrac, Pseudomonasaeru- ginosa, Pseulomonas mirabilis, Moraxella catarrhalis; anaerobic non-clostridian flora: Bacteroidesiradillis, Bacteroides melaninogenicus, Fusobacterium nucleus peptococcus, Veptobacillus peptococcus; fungal flora: Candida pss., Aspergilluss pp.

These microorganisms are sown in monoculture as well as in various associations with each other - mainly aerobic-anaerobic and bacterial-fungal [7, 22].

According to the literature, there are differences in the microflora in different courses of the disease. S. Ziuzio, W. Stepniewier (1980) found bacterial flora predominantly homogeneous in acute sinusitis and mixed in chronic sinusitis.

At the same time, it is important to identify the microflora of the oral cavity and the upper jaw cavity [27, 31].

Studies conducted by A. and Bogatov (1991) show that the immediate cause of chronic periodontitis is often acute and acute periodontitis (up to 50% of cases) or various complications of therapeutic and surgical interventions on teeth (up to 20% of cases).Radical cysts and osteomyelitis of the dental cavities in contact with the floor of the maxillary sinus are found in 13% of cases with almost the same frequency. In his research, A.I. Bogatov (2000) established that the source of infection in the emergency room is often the first mol (56.6% of cases). In a number of cases, premature or poor sanitation of the oral cavity is a prerequisite for the development of VVR.

Currently, there are numerous reports in the literature regarding the development of VCH due to the penetration of filling material into the VCH cavity after endodontic treatment of upper jaw teeth (UJ) [3, 17, 29, 30].

One of the frequent causes of the development of VVR is a cavity perforation, differing in etiology, localization, size, and course. According to a number of authors [12, 16, 35], the development of emergency situations against the backdrop of existing guaranteed communication occurs in 41.2% to 91.7% of cases. There is information in the literature that the most frequent

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group of VCH sinus perforations occurs with the removal of lateral VCH teeth [2, 5, 10]. In the literature, the rarest causes of chronic kidney disease are delayed eruption of third molars, as well as inflammation around retinated and semi-retinated teeth [23]. There are clinical facts indicating the development of VCR after the operation to raise the bottom of the VCHP with sub-intral augmentation [9, 33].

Patho-morphological changes in chronic obstructive pulmonary disease generally correspond to the stages of the inflammatory process: alterative-exudative, productive, and fibrous. Due to the spread of infection from the alveolar ridge of the tooth to the left ventricle, there is mucous-purulent discharge, irreversible morphological changes in the mucous membrane and the formation of necrotic tissues [7, 13].

Later on the ciliary activity of the ciliary epithelium suffers, i.e. it is blocked. Meanwhile, the average number of functioning glands in the mucous membrane is significantly reduced, and general and local cellular immunogenesis is disrupted [22]. In the development of the pathological process against the background of CHD, there is constant irritation of the mucous membrane of the oral cavity with oral fluid, and the mucous membrane begins to react with productive inflammation. This process is often localized, affecting the lining only in the area of the alveolar bucket, it occurs weakly, asymptomatically, therefore it is not always diagnosed.

Factors of a general nature contribute to the development of maxillary sinusitis (MS): a decrease in the body's immunological reactivity, the impact of adverse environmental conditions [13]. The prolonged exposure of microorganisms, products and the vital activity of isodontogenic lesions, as well as autoinfection of the oral cavity, penetrating into the oral cavity, play a certain role in reducing immunological reactivity in the maxillary sinus after its perforation.

Microbial effects on the whole organism and on the already altered, pre-sensitized mucosa of the maxillary sinus from the focus of odontogenic infection determine the further development of the inflammatory process [24]. Chronic granulating and granulomatous periodontitis contribute to the development of odontogenic chronic periodontitis [24, 27].

The bone barrier between the IVC floor and the pathological focus due to inflammation of the bone tissue of the dental alveolus undergoes resorption, in fact, the integrity of the IVC floor is disrupted, which can occur long before the tooth is removed [26, 30, 32]. In the majority of patients with chronic modontogenic perforating VCH with the presence of a persistent fistula at the bottom of the maxillary sinus, thickening and polypoid transformation of the maxillary sinus mucosa are observed [1, 10, 20].

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According to S.Z. Piskunov and co-authors. (2004), productive changes in the VCH membrane in chronic odontogenic sinusitis, unlike rhinogenic ones, are localized in the area of the alveolar cavity and the anterior sinus wall at the initial stage.

It has been established that in odontogenic VCH with puncture of the sinus floor, inflammatory changes of a chronic nature are detected in the mucous membrane, which may be caused by frequent exacerbations, inflammation in the area of the peri-apical lesions of the teeth adjacent to the floor of the VCH. Another important factor influencing the development of VCHP is the size and throughput of the VCHP natural joint.

According to modern concepts, this factor is leading in the pathogenesis of sinusitis [18]. As a result of nasal mucosal edema and VCHP, the permeability of the natural opening of the nasal cavity is disrupted, and the ventilation and drainage function of the nasal cavity is reduced. With complete obturation of the oxygen membrane, due to the absorption of oxygen by the mucous membrane into the VCHP, negative pressure is created, veins are dilated, and stagnation of the mucous membrane occurs. This exacerbates the swelling of the tissue. As a result of pressure drop, hypoxia and hypercapnia, conditions are created for the growth of aerobes and facultative anaerobes [14]. Thus, a vicious circle is formed that determines the outcome of the disease. If it is not torn, irreversible changes develop in the mucous membrane [8, 25, 28].

CONCLUSIONS

Thus, summarizing the above, the following conclusions can be drawn:

Currently, VCH is one of the most common dental diseases, one of the causes of which is a perforation of the upper jaw due to errors in the technique of removal and endodontic treatment of VCH premolaries and molars, and it is a serious general medical problem, as the majority of patients are of working age.

The main etiological factors in the development of VCHD are bacterial and fungal microflora with diseases of the upper jaw teeth (chronic periodontitis, radicular cysts, pubic osteomyelitis, periodontitis), foreign bodies in the VCHD and reconstructive operations on the VCHD, as well as sensitization of the body. Topographic anatomical relationships between VCH and VCHP teeth are the prerequisites for the development of VCHS.

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