Odontogenic causes of maxillary sinusitis and their management

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Abstract
Odontogenic maxillary sinusitis is an uncommon but important disease entity and is often recognised late. The increasing use of computed tomography (CT) in the assessment of unilateral sinus symptoms has improved the ability to recognise and manage this group of conditions. Here we discuss the details of a number of dental causes of maxillary sinus disease. A description of the journey of a tooth is used to demonstrate how certain pathologies occur and how they may lead to disease within this specific region. Management options are also discussed which often require close collaboration with dental and oral/maxillofacial colleagues.


Key words
Maxillary sinus, sinusitis, odontogenic

Introduction
Odontogenic sinusitis accounts for up to 10%-12% of all cases of maxillary sinusitis\(^4\). A simple look at the relevant anatomy explains the relationship, with the floor of the maxillary sinus formed by the alveolar process of the maxilla (Figure 1). The formation and continuing integrity of this alveolar bone is intimately linked to the development and maintenance of normal, healthy dentition.

A wide range of dental problems, both primary and iatrogenic, can compromise the normal functioning of the maxillary sinus. A disruption in the normal mucociliary pathway created by the ciliated pseudostratified columnar epithelium within the maxillary sinus can lead to mucus stasis, overgrowth of organisms and sinus mucosal inflammation\(^5\). There is a two-fold increase in maxillary sinus disease in patients with periodontal disease and this relates to the close proximity of teeth to the maxillary sinus\(^6\). All maxillary teeth have the potential for causing problems in the sinonasal cavities but the first and second permanent molars are those most commonly involved in maxillary sinusitis due to their root morphology and positioning. Less commonly, the maxillary second premolars and third molars can be involved\(^7\).

A dental cause for maxillary sinus disease should be considered in those patients with symptoms of unilateral maxillary sinusitis with a history of odontogenic infection, dento-alveolar surgery or in those patients resistant to conventional sinusitis therapy. The most common cause of odontogenic sinusitis is iatrogenic and this accounts for over half of cases reported in the literature\(^8\).

![Figure 1: Section showing the proximity of tooth roots to floor of maxillary antrum (adapted from Figure 1003 in 'Anatomy of the Human Body' - Henry Gray 1918)](image)
The tooth journey

Knowing the basics of odontogenesis and the journey of a tooth from embryonic cells to an erupted, functional structure will give a greater understanding of the origin and behaviour of odontogenic pathology relevant to the maxillary sinus.

The **tooth germ** is essentially a collection of cells derived from the first pharyngeal arch and neural crest. It is connected to the oral cavity via an in-growth of oral ectoderm – the **dental lamina** and is organised into three main parts:

- **the enamel organ** - gives rise to ameloblasts which produce enamel and also the Hertwig Epithelial Root Sheath which determines the shape of the tooth roots
- **the dental papilla** - produces odontoblasts which form dentine
- **the dental follicle** - produces cementoblasts, osteoblasts and fibroblasts which give rise to cementum, the periodontal ligament and alveolar bone which are the supporting structures of a tooth

Tooth development progresses through various cell production and organisation stages which include the bud, cap and bell stages, then the formation of the dental hard tissues and the formation of the tooth supporting structures. Each of the cell layers and stages of odontogenesis can give insight into the origin of future odontogenic pathology relevant to maxillary sinusitis. In addition to normal odontogenesis, teeth must follow particular eruption pathways to become functioning, healthy oral structures. Impacted teeth are common and can be associated with various pathological processes affecting underlying bone and adjacent structures.

Even when teeth develop normally and erupt into anatomically correct positions, they enter a relatively hostile environment. A host of microorganisms exist in the oral cavity, deleterious to both the calcified tissues of the teeth themselves and the tooth supporting tissues. In addition, a variety of non-microbial threats such as mechanical abrasion/attrition/trauma and chemical agents also exist. The resultant pathology itself can impact upon the maxillary sinus, as can the treatment modalities used by dentists/surgeons to eliminate the pathology.

We discuss the common odontogenic causes of sinusitis below and their relationship to this journey. These can be categorised into three main categories; benign/malignant pathology, infective/inflammatory causes and iatrogenic causes.

A. Benign and malignant pathology

Ectopic teeth

Impacted teeth are relatively common occurrences, especially involving third molars, maxillary canines and maxillary second premolars. Normal eruption is interrupted or impeded resulting in abnormal tooth positioning. This is especially true for late erupting teeth or those with long eruption pathways, such as the maxillary canine. Impacted teeth can remain dormant and cause no problems but any associated cystic change or surgical treatment to remove them can result in maxillary sinus disease.

Ectopic teeth/supernumeraries/odontomes are only seen rarely in non-dentate areas such as the maxillary sinus. They are commonly only identified during imaging for investigation into the cause for any consequential maxillary sinusitis. When secondary pathology exists, early surgical intervention is recommended for removal of the tooth and any associated cyst which may require open or combined endonasal and oral approaches.

Odontogenic cysts

The lining of these cysts are all derived from the remnants of the tooth-forming organ and can be subdivided into developmental and inflammatory types. These lesions can cause significant bony destruction and the tissues surrounding them can become inflamed/infectected and lead to secondary maxillary sinusitis.

Radicular cyst

- The most common odontogenic cyst, accounting for more than 65% of all such lesions
• Always develop within granulomas in the periapical tissues of non-vital teeth (but not all periapical granulomas progress to cysts).

• Lining is derived from the rests of Malassez which are groups of cells left over from Hertwig’s epithelial root sheath and is supported by a chronically inflamed fibrous capsule.

• Enlarge slowly and generally do not grow to very large dimensions (Figures 2 and 3).

• Treatment involves either endodontic therapy or removal of the non-vital tooth and enucleation of the cyst lining.

**Dentigerous cyst**

• Originate in the follicular tissues overlying the crown of unerupted teeth

• Lining is supported by a fibrous capsule free from inflammatory cell infiltration.

• True dentigerous cysts are attached to the amelocemental junction of the tooth.

• Most commonly associated with late erupting teeth, such as third molars, second premolars and upper canines.

• Can become very large, causing significant bony resorption.

• Reports of mucoepidermoid carcinoma are associated with dentigerous cysts but this is extremely rare⁶

• Simple enucleation of the cyst lining along with removal of the impacted tooth is usually curative in the case of dentigerous cysts, although studies have shown good success rates with surgical decompression techniques⁷

**Keratocystic odontogenic tumour (KCOT):**

• Arise from the remnants of the dental lamina

• Locally aggressive growth pattern (hence the terminology of tumour)

• Tendency to recur due to friable nature of the cyst wall and tendency to advance with finger-like projections into surrounding bone, therefore surgical curettage alone is often inadequate

• Can reach large sizes with few symptoms and less bony deformity than other cysts due to antero-posterior expansion

• Radiologically can have a multi-loculated appearance with resorption of associated teeth roots

• Multiple keratocysts can be associated with Gorlin syndrome (Basal Cell Nevus Syndrome)

• KCOT’s require a more aggressive surgical approach to ensure clearance.

**Invasive odontogenic lesions**

These are rare lesions, the most common of which is the ameloblastoma.

**Ameloblastoma**

• Benign but locally invasive tumour of odontogenic epithelium.

• Majority occur in the mandible but those which do arise in the maxilla can often involve the maxillary sinus.

• Generally slow growing and asymptomatic in the early stages.

• Can cause significant bony expansion, tooth resorption and increasing facial deformity.

• Radiographically they are multi-loculated in appearance, although a separate unicystic ameloblastoma exists that is more amenable to conservative surgical treatment.

• Normal ameloblastomas have a high recurrence rate following simple curettage. Due to islands of tumour infiltrating cancellous marrow spaces.

Most other benign odontogenic tumours, except perhaps the odontogenic myxoma, follow a less aggressive pattern than the ameloblastoma.

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**Figure 3: Note how left contralateral tooth does not cause significant sinus disease despite root penetration into maxillary sinus**
Malignant odontogenic tumours, according to the WHO classification, consist of:
a. Malignant Ameloblastoma
b. Primary Intraosseous Carcinoma
c. Other carcinoma arising from odontogenic epithelium, including from odontogenic cysts

These are rare lesions but again have potential to impact on the maxillary sinus.

B. Infective and inflammatory causes
Oral infective/inflammatory causes of maxillary sinusitis include dental periapical pathology, advanced periodontal disease, secondary infection of (non-inflammatory) odontogenic cysts and rarely Actinomycosis, a subacute/chronic bacterial infection usually caused by Actinomyces israelii.

Periapical pathology
In a non-vital tooth, either as a result of dental caries or trauma, the necrotic pulp tissue loses its ability to counter invasion by microorganisms from the oral cavity. As a result the root canal space becomes colonised by microbial communities which initiate and sustain inflammatory processes in the tissues surrounding the tooth roots. This can lead to tissue necrosis and abscess formation.

Periodontal disease
In periodontal disease the initial site of bacterial colonisation is the gingival sulcus (the space between the tooth and gingiva) rather than within the root canal system. The inflammatory process begins as gingivitis which is not associated with alveolar bone loss but depending on a number of complex factors, this can progress to periodontal disease which causes bony resorption and apical migration of the disease process.

The thickness of the maxillary floor may influence the chance of inflammatory tooth disease leading to ascending sinus inflammation. Direct communication between the odontogenic (or otherwise) inflammatory process and the sinus lining influences the mucociliary pathways within the maxillary antrum, leading to mucus stasis, bacterial colonisation and a proliferating inflammatory process. Most infective cases involve a polymicrobial aerobic-anaerobic infection with gram-negative anaerobes particularly prominent. Other cases involve anaerobic streptococci, gram-negative bacilli, and Enterobacteriaceae.

C. Iatrogenic causes
The other main causes of sinus pathology are those related to dental procedures and their associated complications, accounting for more than 60% of cases in the literature. Any change in the normal anatomy of the teeth and the surrounding structures can lead to a greater propensity for consequential sinus disease.

Extractions
These can sometimes lead to an oro-antral communication (OAC), allowing material and organisms from the oral cavity to enter the maxillary sinus. The close proximity of the roots to the sinus and the morphology of certain multi-rooted teeth can mean that very little or no bone remains after a tooth is extracted. Sometimes an OAC is inevitable (Figure 4) and unavoidable but there are surgical techniques to help minimise the risk.

Small OACs may close spontaneously but any communication can develop into an oro-antral fistula (a pathological epithelial-lined communication between the sinus and oral cavity) especially in the presence of underlying sinus pathology. It is important the surgeon takes note of any communication and initiates appropriate management. This can consist of conservative management of small defects (antibiotics, decongestants, avoidance of sharp air pressure changes within the sinus) and a variety of surgical techniques to primarily close larger communications. It is important to excise the epithelial lining of established fistula tracts prior to surgical closure. Techniques commonly involve local soft tissue flaps (buccal advancement/palatal rotation flaps) sometimes in conjunction with the buccal fat pad (Figure 5-7). A variety of bone grafts, pedicled tongue flaps, resorbable and non-resorbable membranes, bone substitutes and different combinations of these have all been used with varying degrees of success to close such defects. Treatment by the endoscopic approach has also been described.

In addition to OACs, the roots of teeth can be displaced into the antrum during oral surgical procedures. In some instances this may result in no associated sinus pathology
but the majority require surgical removal. Simple radiographs can be helpful in locating these displaced fragments but often CT or cone beam CT imaging is required. Surgical approaches to retrieve displaced roots can be trans-alveolar or via a Caldwell-Luc approach intra- or via an endonasal approach which requires a wide antrostomy to gain access (Figures 8 and 9).

**Implants**

Dental implants require osseo-integration within sufficient healthy bone to be successful. Insufficient planning and poor surgical technique can lead to protrusion of a dental implant into the maxillary sinus and this in turn can act as a nidus of infection. The placement of dental implants is increasingly being facilitated by sinus-lift procedures. These involve different approaches to access and lift the Schneiderian membrane lining of the sinus and to encourage the growth of new alveolar bone, allowing placement of the implant. The use of osseo-conductive bone substitutes and (usually) resorbable collagen membranes often form part of this surgical procedure. Such surgical techniques in themselves carry the risk of initiating and establishing inflammatory sinus pathology, especially if the Schneiderian membrane is perforated and they do not completely prevent the potential for secondary sinusitis\textsuperscript{13}.

There is evidence however, that penetration of a dental implant into the maxillary sinus with membrane perforation does not have long term clinical or radiological sequelae especially if penetration is less than 3 mm\textsuperscript{13}. There is on-going discussion regarding the use of prophylactic antibiotics for surgical dental implant procedures and currently their use is recommended\textsuperscript{15}.

**Root canal therapy**

Conventional root canal therapy involves the chemico-mechanical preparation of the root canal system of a tooth to remove necrotic pulpal debris and reduce the microbial load within. This is followed by three dimensional obturation and sealing of the canals to prevent residual bacteria releasing products into the surrounding periapical tissues. The root filling material gutta-percha is commonly used to obturate the resultant open canal following
preparation. Extrusion of materials/chemicals/instruments used during root canal therapy beyond apex of tooth into sinus is possible. Whilst the obturating material itself is inert and unlikely to cause much inflammation, irritants such as sodium hypochlorite which are used in root canal therapy before application of the gutta percha could be forced into the maxillary sinus. This can lead to localised necrosis, pain and infection.

Foreign bodies
A variety of foreign bodies/materials can enter the maxillary sinus via the oral cavity. These include the aforementioned root filling materials, irrigant solutions, fractured instruments, dental implants/components and socket-packing materials. They can facilitate the passage of oral microbes into the sinus and the foreign body can impede the normal mucociliary clearance pathways, cause a localised inflammatory reaction and lead to acute or chronic maxillary sinusitis. If the object is confined to the sinus itself then a wide antrostomy and retrieval may be possible but sometimes these require an oral, combined and rarely an open approach.

Conclusions
The majority of causes for odontogenic maxillary sinusitis are secondary to dentally related procedures. Occasionally a primary lesion of the teeth may lead to a source of maxillary sinusitis. Radiological investigations are increasingly identifying the causes of this disease entity and aid with the planning for surgical management, particularly for the approach required. A broad awareness of local anatomy and pathology with a multidisciplinary team approach to treatment is the best way to successfully manage this potentially refractory type of sinusitis.