Review article

Odontogenic maxillary sinusitis: A comprehensive review

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Abstract Odontogenic maxillary sinusitis (OMS) is a well-recognized condition in both the dental and otolaryngology communities. Close to 30% of cases of unilateral maxillary sinusitis may have an underlying dental pathology. Failure to identify a dental cause usually lead to cases recalcitrant sinusitis often associated with serious complications. The aim of this study is to describe the literature findings on odontogenic maxillary sinusitis that discuss anatomy, epidemiology, etiology, bacteriology, diagnosis and treatment.

The present review is based on a current search using bibliographic database and academic search engine. All the articles on odontogenic maxillary sinusitis published after 2000 were included. This study seeks to provide clinicians with evidence that motivates a comprehensive approach to the evaluation and management of OMS. Controversies on diagnosis and management have been addressed and data from different treatment plans were collected by exploring relevant publications.

The surgical treatment of OMS is based essentially on the dental surgery, combined with endoscopic sinus surgery, in order to completely remove the infection, restore the physiological drainage of the sinus and prevent recurrences of sinusitis. A multidisciplinary otolaryngology and dental team is mandatory to successfully manage the dental pathology and the complications resulting from the dental treatments.

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Introduction

Maxillary sinusitis (acute or chronic) is defined as a symptomatic inflammation of the maxillary sinus, usually caused by viral, bacterial, allergic or fungal rhinitis.[1] However, any disease arising from dental or dentoalveolar structures could damage the floor of the maxillary sinus leading to a sinusitis known as odontogenic maxillary sinusitis (OMS). The OMS is a well-recognized but understudied form of sinusitis that requires a unique treatment regimen that differs from non-odontogenic sinusitis.[2]

This review aims to provide better insight into various dental etiologic factors of OMS, to clarify diagnostic disease characteristics and present different treatment plans. For this reason, a bibliographic search between January 2018 and January 2019 was performed in PubMed, Scopus, Cochrane Library, Web of Science and Science Direct and only articles published after 2000 were considered. Despite not using a systematic methodology in this review, the free term investigated was odontogenic sinusitis. All the relevant articles discussing anatomy, epidemiology, etiology, bacteriology, diagnosis and treatment were included. To make the present review more didactic, the findings are presented according to the topics mentioned in the previous paragraph.

Anatomy

The maxillary sinus is the first of the paranasal sinuses to develop in human foetal life. The maxillary sinus reaches its full development with the eruption of permanent teeth between 12 and 14 years of age, containing an average volume of 15—20 mL.[2] The roots of the maxillary second molars are in closest proximity to the sinus floor, followed in frequency by the roots of the first molar, third molar, second premolar, and first premolar.

The floors of the maxillary sinus are consisted of thick cortical bone, not allowing a direct penetration of odontogenic infections into the maxilla bone. However, the alveolar bone of the maxilla can become thinner with increasing age, leaving a layer of mucoperiosteum with respiratory epithelium between the maxillary sinus and the oral cavity, the Schneiderian membrane.[1,3] When the pneumatization of the sinus continues even after the eruption of the permanent teeth, the third molar, premolars and canine teeth may protruded into the sinus.[3]

Epidemiology

Before the 1970s, approximately 10%—12% of OMS cases have been attributed to odontogenic infections.[4] According to Maillet et al.,[5] the incidence can raise to 51.8%. Patel and Ferguson[6] have reported that close to 30% of cases of unilateral maxillary sinusitis may have an underlying dental pathology. With the frequent use of sinus endoscopic surgery, an increased incidence of OMS has been recorded over the last decade.[7]

Etiology

The most common etiology of OMS is dentoalveolar surgery or odontogenic infection with perforation of the Schneiderian membrane.[8] Troeltzsch et al.[9] demonstrated that dentoalveolar surgical intervention with subsequent oroantral fistula (OAF) formation was the main cause of OMS. However, Ferguson[1] supported that the cause of OMS is usually periapical or periodontal infection defined as infections around the teeth. A recent systematic review[10] studying the causes of OMS from January 1980 to January 2013, among 674 patients, showed that iatrogenic etiology accounted for 65.7% of cases, apical periodontal pathologies for 25.1% (apical periodontitis — 16%, apical granulomas — 5%, odontogenic cysts — 2.5%), and marginal periodontitis for 8.3%.; iatrogenic causes included impacted tooth after dental care, artificial implants, dental amalgams and OAF. In the same study,[10] the maxillary teeth most affected were, in order of frequency, the first molar (35.6%), second molar (22%), third molar (17.4%), and second premolar (14.4%). In a more recent systematic review, Akhlaghi et al.[11] demonstrated that OAF, as a complication of tooth extraction, was the most common cause of OMS among all dental etiologies. Table 1 shows the main etiologic factors involved in the pathogenesis of OMS according to different studies.

Odontogenic infections begin with the attachment of bacteria to the outer surface of teeth, eventually breaking down the outer enamel and inner dentin and making its way into the vital pulp.[25] Once the infection enters the pulp, it leads to the necrosis and pus formation. The body is unable to eliminate the source of infection because the necrotic pulp is protected within the tooth roots. Bacteria colonize the apical portion of the root and their toxins can damage tissues causing a periapical infection. An acute, rapidly-spreading infection is much more destructive than slowly-developing inflammation, affecting the adjacent maxillary sinus in a short time.[26] Bacteria from the lesion can spread to the adjacent tissues and activate a reaction from the Schneiderian membrane epithelium which seems to be hypertrophic and inflamed. If the endodontic treatment fails and microorganisms resist and continue growing, the formation of secondary periapical lesion will be noticed.[26]

Extrusion of dental materials used in root canal therapy into the maxillary sinus has also a high risk of producing OMS. According to a meta-analysis[27] based on articles published between 1986 and 2007, iatrogenic causes (55.9%) for OMS included extrusion of endodontic obturation materials into the maxillary sinus (22.7%), such as amalgam after apicoectomies (5.3%). Other endodontic microtools and root filling materials such as gutta-percha cones can also penetrate the apical foramen protruding into the maxillary sinus floor and resulting in OMC.[28]

Less commonly, dentigerous cysts may be associated with a displaced maxillary molar[29] or an ectopic impacted third molar[30] in the maxillary sinus presenting as OMS. Dentigerous cysts are odontogenic cysts that can occur in the maxillary bone close to the sinus[1,30] and consist of a fluid-filled sac with a thick surrounding; that cyst can cause

Table 1　Main etiologic factors involved in the pathogenesis of odontogenic maxillary sinusitis (OMS).

Author	Nb (patients)	Age (y) median/range	Etiology
Mattos et al.[12]	43	53	Prior dental procedures, OAF
Lee & Lee[13]	27	42.9	Dental implant & dental extraction complications, dentigerous & radicular cysts, dental caries
Felisati et al.[14]	257	51.5	Dental implant dislocation, OMS after sinus lift
Andric et al.[15]	14	NR	OAF after tooth extraction
Chemli et al.[16]	22	39	Periapical infection, foreign bodies, cysts, OAC
Fadda et al.[17]	31	51.3	Dental-related bacterial or fungal OMS, OAF after sinus lift, dental implant dislocation
Hoskison et al.[7]	26	46.2	Periapical infection, OAF, foreign bodies
Lechien et al.[10]	674	45.6	Iatrogenic, apical periodontitis, apical granuloma, odontoma, dental implant, foreign bodies
Lopatin et al.[18]	70	16–62	OAF, foreign bodies
Costa et al.[19]	17	NR	OAF, odontogenic cysts, periimplantitis, foreign bodies
Longhini & Ferguson[20]	21	53	Prior dental procedures
Selmani & Ashammakhi[21]	15	45	Foreign bodies
Jiam et al.[22]	9	63	OMS after sinus lift
Kim et al.[23]	19	54.5	Dental implant-related OMS
Chen et al.[24]	18	53.1	Dental implant-related OMS

OAF: oroantral fistula, OAC: oroantral communication, OMS: odontogenic maxillary sinusitis, NR: not reported.
Foreign bodies: roots, tooth, broken instruments, dental amalgam, dental caries.

thinning of the bone under mass effect and, if untreated, it can expand into the maxillary sinus.[29]

The dental implant is increasingly used nowadays for partial or complete edentulous patients; moreover, before dental implantation, in order to increase alveolar bone height, an osteotomy of the lateral maxillary sinus wall is performed following by graft placement, a procedure called sinus lift.[22,24] However, during or after these procedures local infections (peri-implantitis) may cause OMS, which may lead to implant failure or severe complications, such as orbital cellulitis, extradural and subdural infections, and osteomyelitis.[22–24] The dislocation of a dental implant into the maxillary sinus may be another cause of OMS; this is often related to the low density of the maxillary bone and when no sinus lift was performed. Galindo-Moreno et al.[31] reported that 13% of 14 patients with dislocation of dental implant into the sinus presented with acute OMS. On the other hand, Jung et al.[32] evaluated 9 patients with dental implants that had been penetrating the mucosa of the maxillary sinus floor more than 4 mm and in a follow-up of 10 months no clinical signs of OMS were reported.

Oroantral communication (OAC) and epithelialized formation of OAF are common complications during dental surgery which can lead to sinus infection. The OAC can occur with frequencies between 0.31% and 4.7% after the extraction of upper teeth[33]; a displacement of dental root into the sinus may also occur following root fracture.[1,11] During tooth extraction, significant forces are exerted on the alveolar bone, especially in the presence of impacted tooth or widely divergent roots, which makes this procedure more difficult[4]; when a periapical cyst, granuloma or periapical infection is present or during apicoectomy, the surrounding bone becomes eroded and the bone separating maxillary sinus from the oral cavity thinner, resulting in exposure of the sinus.[4,27]

Bacteriology

Odontogenic sinusitis is a polymicrobial infection in which bacteria from both oral cavity and upper respiratory system are involved, in predominance anaerobic species. Brook[34] in 48 patients suffering from OMS, showed that gram-negative bacilli, such as Peptostreptococcus spp. and Fusobacterium spp. were the main anaerobes bacteria, which predominated over aerobes in both acute and chronic OMS. The same author[35] compared the microbiology of aspirates from 5 periapical abscesses of the maxilla and their corresponding maxillary sinusitis (through the inferior meatal antrostomy); they found concordance between periapical abscess and the maxillary sinus flora (mainly anaerobes) in all the patients, underlying the direct extension of such microorganisms from the maxillary molar teeth to the proximal floor of the maxillary sinus.

Puglisi et al.[36] demonstrated that mixed aerobic–anaerobic infections were found in 75% of 12 patients with chronic OMS (main aerobes: *Staphylococcus aureus*, *Streptococcus pneumoniae*, main anaerobes: Prevotella spp., Peptostreptococcus spp.). Bacteria strongly associated with chronic rhinosinusitis like *Haemophilus influenza* and *Moraxella catarrhalis* were not found in OMS.[36] Tashieri et al.[26] showed that apical lesions are mainly caused by Actinomyces spp., an anaerobic bacteria, which avoid the phagocytosis, and is related to the formation of biofilms on root surfaces or the presence of other endoantral foreign bodies.[37] Finally, it has been concluded[11] that anaerobic bacteriological flora is the most common cause of chronic OMS, while the main flora is mixed in patients with acute OMS.

It has been reported that dental roots following endodontic treatment, such as zinc oxide-eugenol, in close proximity to maxillary sinus may result in positive cultures for Aspergillus.[38,39] In Zirk et al. study[40] that included 121

patients suffering from OMS who underwent surgery, aspergillus was found in 5 patients in the sinus, and in 4 cases of aspergillosis a foreign body was detected in the sinus; in addition, aspergillosis was detected once after tooth extractions.

Symptoms

The main symptoms related to OMS are facial pain or pressure, nasal congestion, purulent rhinorrhea that may be unilateral, cacosmia, and postnasal drip. Hoskison et al.[7] reported that 21 (81%) and 19 (73%) out of 26 patients with OMS complained of rhinorrhea and cacosmia, respectively. In a study[13] that included 27 patients with OMS, rhinorrhea was found in 66.7% of cases, cheek pain in 33.3% and cacosmia in 25.9%.

However, these symptoms do not distinguish OMS from other causes of sinusitis, as some patients experience sinusitis-like symptoms, such as dental pain and nasal congestion, whereas others present with minimal sinusitis symptoms and dental pain, because the osteomeatal complex is not obstructed and allows drainage and relief of pressure.[4,6,40-42] Longhini and Ferguson[20] reported that dentists did not diagnose dental infection causing OMS in 6 (85%) out of 7 cases; similarly, 56 (55%) out of 99 of OMS cases were missed on routine dental examination including dental X-ray.[43]

Less than half of patients presenting with OMS report a recent dental procedure[42]; this is because, for example, OMS can appear within 1 year after augmentative dental surgery following graft infection. OMS may also occur after a latency period of almost 4 years as a late complication of dental implantology due to progressive peri-implantitis.[9]

Diagnosis

The diagnosis of OMS should be based on a thorough dental and medical examination, including evaluation of patient's symptoms and past medical history. Patients with history of extractions of the maxilla molars or an endodontic therapy may have OMS.

Clinical examination includes inspection of the buccal mucosa and vestibule for swelling or erythema. In addition, the pulp is tested by using electric or thermal pulp vitality testing, percussion, and palpation in order to determine if the tooth is hale. If there are teeth with existing root canal therapy, the dentist should examine for any untreated or sub-optimally filled root canals, inappropriate core restorations or leaking coronal restorations.[42] The maxilla sinus itself can also be evaluated with intranasal examination with anterior rhinoscopy or flexible nasolaryngoscopy.

Both OAC and OAF can most of the times be diagnosed clinically, with the Valsalva test or by examining the extraction region with a blunt probe.[44] Presence of OAF appears as an altering of the voice due to air leaking from the nose or raising fluid from the mouth to the nose. Additionally, in many cases a small amount of purulent discharge may drip through the OAF.

Radiographic imaging is an essential tool for the diagnosis and management of OMS. Periapical and panoramic radiography are helpful to determine the size of periapical lesions, visualization of pseudocysts, displaced roots, teeth or foreign bodies inside the maxillary sinus.[45] However, panoramic radiography has lower sensitivity than periapical radiography for detection of apical periodontitis due to anatomical superimposition.[46]

Both these diagnostic tests are part of two-dimensional imaging and therefore they are not specific enough to clearly evaluate upper maxillary areas compared to 3D techniques, such as computed tomography (CT).[42,45,47] Moreover, it has been reported that dental radiographs may miss between 55% and 86% of dental pathology that are involved in OMS.[20,43] The CT is considered the gold standard for adequate maxillary sinus imaging because of its high resolution and ability to discern bone and soft tissue and then to detect sinonasal inflammation. Axial and coronal sinus CT views can show the relationship of a periapical abscess with sinus floor defect and determine the exact location of a foreign body within the maxillary sinus.[48-50] Care should be taken during CT examination to include maxillary tooth roots and the floor of the maxillary sinus in all CT studies of the sinuses.[6]

New advances in 3D imaging systems have introduced cone beam computed tomography (CBCT). The CBCT consumes approximately 10% of the radiation dose of conventional thin-slice CT, focusing on image bony details, although soft tissue quality is reduced.[6,51] Compared to panoramic dental radiograph, radiation dosage for CBCT is approximately 10-fold higher;[52] however, the CBCT is actually preferred in the field of implant dentistry, in order to assess the thickness of the floor of the maxillary sinus prior to implantation.[6]

Periapical lesions are better detected by CBCT compared to periapical radiography,[42,51] which can only spot approximately 40% of apical periodontitis on posterior maxillary teeth and 3% of all apical infections extending on the sinuses[45]; however, according to a recent review,[47] the efficacy of CBCT as diagnostic imaging method for periapical lesions is questionable, as CBCT findings have not been correlated with biopsy and histological examination of periapical tissues.

It has been shown that the majority of unilateral maxillary sinusitis cases (more than 70%) are of odontogenic origin.[9,13,53] OMS has most commonly been identified on CT radiographs (axial, coronal planes) as mucosal thickening ≥ 2 mm of the maxillary sinus membrane, associated with a dental focus responsible for sinus pathology, such as OAC, foreign bodies (dental fillings, teeth roots), periapical abscess — granulomas, or extraction site (Fig. 1).[54-57] Vidal et al.[41] supported that the mucosal thickening could be limited to the area of a tooth presenting one or more of the following pathological findings: caries, defective restoration, periapical lesion or an extraction site. Mehra and Jeong[2] have extensively described the process transition from endodontic pulpal necrosis to periapical osteitis and finally to periapical abscess that is radiographically detected. Pokorny and Tataryn[55] found that approximately two-thirds (64%) of the OMS cases (in total 33 patients) showed evident periapical—periradicular infection on CT imaging; in the remaining 36% there was no obvious dental disease on CT, including cases with failure of root canal therapy and loss of bone. The latter was characterized by absence of the bony partition between the root apices and
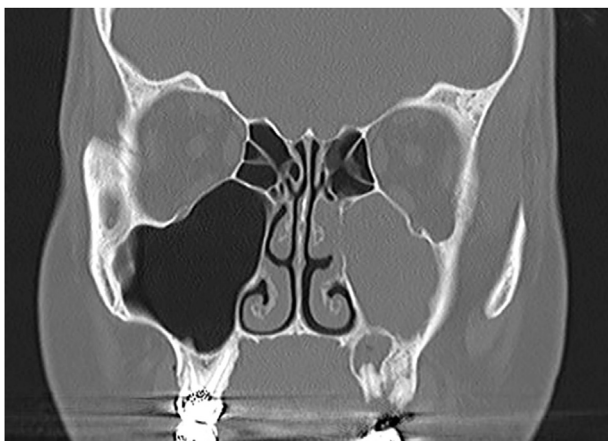
Figure 1 Periapical abscess on the 1st upper left molar tooth with associated complete opacification of the left maxillary sinus and obliterating the left osteomeatal complex.

the sinus mucosa, or infected tooth roots that protrude through the maxillary sinus cortical floor, eliciting inflammatory changes directly into the sinus.

Treatment

Appropriate antibiotic therapy for OMS should initially be given targeting aerobe and anaerobe bacteria. For this reason, amoxicillin combined with clavulanate, which is a beta lactamase inhibitor, is preferred.[40,41,58] Zirk et al.[40] reviewed 121 patients suffering from OMS and found the highest susceptibility rates with piperacillin (93.9%), cefotaxime (78.1%), cefuroxime (69.4%) and clindamycin (50%); fluoroquinolones, such as moxifloxacin (86.2%) and ciprofloxacin (62.2%), and tetracyclines (62.9%) could be alternative medication in patients with confirmed penicillin allergy.

A specific management protocol is not yet established for further management of OMS. It has been demonstrated that both dental surgery and endoscopic sinus surgery (ESS) have shown excellent results, but the ideal sequence of management and time has not been presented yet.[59,60] In case where there is clear evidence of dental infection source, our treatment should address first the dental pathology.[58] This may involve endodontic treatment with root canal, apicoectomy, or dental extraction.[42] Endodontic treatment involves removal of the neurovascular tissue within a tooth (dental pulp) and obturation of the empty canal space with a synthetic material, such as gutta-percha cones. If root canal therapy is unsuccessful, it is advisable that the offending tooth is extracted.[1] Extraction of maxillary posterior teeth must be done carefully to avoid OAC or even displacement of root tips into the maxillary sinus.[44]

If symptoms persist, the ESS is recommended, especially if the osteomeatal complex is blocked[12] and the height of the thickened mucosa is more than one-half of the maxillary sinus.[24] In contrast to ESS, the classical Caldwell-Luc remains an oroantral procedure with a higher complications rate, intraoperative (bleeding, facial swelling, infraorbital nerve damage) and long-term (OAF, teeth devitalization, facial paresthesia) complications.[44]

Many authors supported that the concomitant management of ESS and dental surgery was the most effective, ensuring complete resolution of the infection and preventing recurrences and complications.[13,14,61] A study[7] in UK found that the most common management for OMS was synchronous ESS and dental surgery. However, Wang et al.[59] reported that 33% of their patients with successfully resolved OMS underwent sinus surgery alone, underlying that exclusive ESS could be an effective treatment approach. These discrepancies sometimes depend on the grade of severity of both dental disease and sinusitis, as patients that have cured with dental surgery may have a lower sinonasal disease burden, whereas other suffering from minimal dental disease may completely recover after ESS alone.[58] In any case, dental surgery should be the core component of management,[10] and this was well demonstrated by Longhini and Ferguson,[20] showing that 6 (29%) patients who had only undergone ESS failed to recover from OMS until dental surgery was performed. Finally, Fadda et al.[17] have recently suggested the combined approach (oral and sinonasal route) in the following indications: i) removal of infected dental implants with apical portions penetrating into the maxillary sinus or any other migrated material, ii) removal of infected grafting material which can be more difficult to eliminate with endoscopy, iii) foreign bodies or odontogenic cyst, iv) periapical odontogenic infections of the teeth, v) maxillary osteitis or osteomyelitis, vi) dentigerous cysts, vii) dental extraction-related complications, and viii) the closure of OAC.

Dental implant-related OMS should initially be treated with antibiotics, such as amoxicilline-clavulate associated with clindamycin,[22] or fourth-generation quinolones which cover anaerobes.[23,61] If OMS persists, an ESS is indicated, consisted in wide middle antrostomy to allow the removal of inflammatory mucosa, granulation tissue or foreign bodies located in the maxillary sinus; lesions in the ethmoid, frontal and sphenoid sinuses were equally eliminated, depended on the diseased sinus.[22,23,61]

Discussion

The present study proposed to present an update based on recently published researches that explore OMS. The close anatomic proximity of the root apexes of the teeth (mainly second molar) to the maxillary sinuses makes dental disease a potential source for spread of this infection into the maxillary sinuses.

The incidence of OMS is very low despite the high frequency of dental infections[4,13]; however, this incidence appears to be increasing.[7] Odontogenic sinusitis is often refractory to initial antibiotic therapy due to the polymicrobial, anaerobe-predominant nature of this disease. OMS should always be considered by otolaryngologists and dentists when a patient has unilateral nasal symptoms that do not respond to medical treatment, mainly after a recent history of maxillary dental procedure.[53,55,59] Usually, OMS may follow periapical or periodontal infection, endodontic lesions, OAC—OAF or dental implant infection (Table 1).

The diagnosis of OMS requires systematic dental examination and CT tomography, including 3D techniques, such as cone beam computed tomography.[6,42] Axial, coronal and

sagittal CT slices of the maxillary sinus can reveal dental foci, such as periapical abscess (Fig. 1), foreign bodies, inflammatory cysts, periapical granuloma, and OAC—OAF.[45,47,57]

ESS should be performed on patients who fail initial antibiotic therapy and dental treatment. However, a common consensus on the treatment plan of OMS is not yet established; there are studies that have reported successful results with ESS and dental surgery, although the ideal sequence of management remains unclear. ESS is strongly recommended when the osteomeatal complex is blocked[12] and the height of the thickened mucosa is more than one-half of the maxillary sinus.[24] Moreover, the dental source of the infection (root canal therapy of causative tooth, periapical or periodontal infection etc.) must be eliminated in order to prevent recurrence of sinusitis. Most recent studies have supported that the concomitant management of ESS and dental surgery was the most effective, ensuring complete resolution of the infection.[10,12,17,24,58—61]

We recommend antibiotics as first line treatment for dental implant-related OMS; if OMS persists, ESS is indicated. Intraoral intervention is not necessary except for the presence of OAC, a local buccal or palatal flap could then be performed.[14,23,24] In case of OMS, it is not clear whether the dental implant should be removed or not. According to a treatment protocol, Felisati et al.[14] proposed combined approach with removal of the implant dislocated into the sinus, while other authors[23,24] preserve the implant, even after sinus lift[22]; additionally, Chen et al.[24] suggested that dental implants should be removed if they are mobile or present with severe peri-implantitis.

Since dental implantation or maxillary sinus lift may involve OMS, these procedures should always be preceded by otolaryngological examination, as the success rate of dental implants depends on the status of the osteomeatal complex of the maxillary sinus[23] and so does the occurrence of OMS after the sinus lift.[62]

Finally, according to the literature, the management of OAC depends on the size of defect, time of diagnosis and presence of OMS.[63] Cases of OAC, if are present for more than 3 weeks, should be surgically closed and ESS is required to eliminate granulation tissue and to keep osteomeatal complex patent[64]; absence of sinus disease is extremely important to achieve OAC closure.[58] If OAC is smaller than 3 mm and without epithelization it generally closes spontaneously in the absence of infection.[63,64] When OAC is larger than 3 mm surgical closure is indicated with buccal advancement flaps; palatal flaps are recommended for large bony defects.[65] Autogenous grafts derived from chin, retromolar area or iliac crest can be used when the soft tissue flaps fail or a chronic OAF exists.[63]

Declaration of Competing Interest

The authors have no conflicts of interest relevant to this article.

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