

# Possible disease transmission by contaminated mouthguards in two young football players

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Previous studies have demonstrated that athletic mouthguards worn by ice hockey and football players harbor large numbers of bacteria, yeasts, and molds, some of which are either opportunistic or frank pathogens. This article details the clinical history of two junior high school football players. The first player had cellulitis of the leg after a non-break injury. The same unusual bacterium was isolated from both the athletic mouthguard and abscess cultures from the wound. The second patient suffered an attack

of exercise-induced asthma so severe that his inhaler could not control the symptoms enough for him to resume play. This child's mouthguard was contaminated with five different species of mold. The clinical implications of mouthguard contamination, possible avenues of disease transmission, and recommendations for mouthguard care are discussed.

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The vulnerability of teeth to trauma from contact sports (such as football and ice hockey) has underscored the protection provided to the dentition by athletic mouthguards.<sup>1-7</sup> The ADA also has recommended mouthguards for all contact sports.<sup>8</sup> Football and ice hockey players at all levels have been the most compliant in wearing mouthguards, to the extent that failure to do so can result in a team penalty.<sup>9-12</sup>

Recent studies of mouthguards retrieved from football and ice hockey players found that these devices harbor a wide range of opportunistic or frank pathogenic bacteria as well as fungi, including both yeasts and molds.<sup>13,14</sup> When these mouthguards were examined by scanning electron microscopy (SEM), the matrices were found to be equally porous to dentures, as reported in the literature (Fig. 1).<sup>15-19</sup> Microorganisms were found both on the surfaces and in the porosities of the used mouthguards; these microorganisms often were incorporated into well-established, complex

biofilms (Fig. 2).

These studies also demonstrated that used mouthguards have sharp and jagged edges capable of lacerating the oral tissues. This means that the mouthguard microorganisms have direct access to the rich vascular system of the oral cavity; in addition, the acts of swallowing and mouth-breathing during mouthguard wear provide microorganisms with direct access to the gastrointestinal and respiratory tracts.

Recent studies have compared the spectrum of microorganisms found in mouthguards worn by football and ice hockey players to those found in mouthguards worn by medical student volunteers.<sup>14</sup> In general, medical student mouthguards had fewer potentially pathogenic microorganisms than those worn by either football or ice hockey players.

Mouthguards worn by football players had large quantities of soil microorganisms such as *Bacillus cereus*, *Rhodotorula* spp. (yeasts), and molds. Ice hockey players' mouthguards were heavily contami-

nated with Gram-negative bacteria (especially *Pseudomonas* spp.) and yeasts such as *Candida* spp.

Several articles further underscore the unique types of infectious diseases associated with athletes. One article documented the problems that football players, wrestlers, and fencers experienced with methicillin-resistant *Staphylococcus aureus* (MRSA).<sup>20</sup> An equally disturbing article described life-threatening bacterial meningitis affecting two New Jersey high school students whose only connection was that they both played ice hockey for the same team.<sup>21</sup> Daum and Herold pointed out that the MRSA problem has increased 25-fold in children, especially boys "with no identified risks."<sup>22</sup> A 2002 study involving football and ice hockey players found *S. aureus* in 31% of the football players' mouthguards and 47% of the ice hockey players' mouthguards, compared to only 14% of the medical student mouthguards.<sup>14</sup>

Although opportunistic and pathogenic microorganisms have

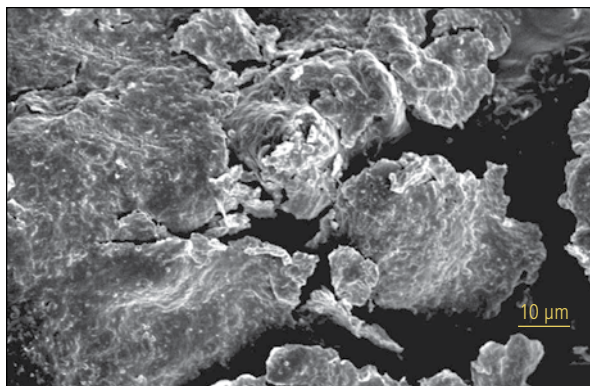


Fig. 1. An example of a football mouthguard that has been boiled but not worn. Note the large defects in the surface that would allow for microorganism penetration. (Original magnification: 1,000x.)

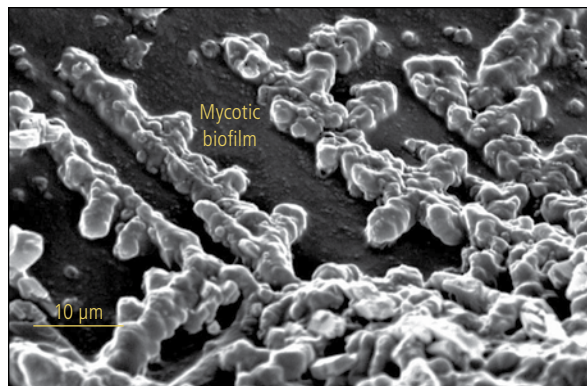


Fig. 2. A mouthguard that has been both boiled and worn. Note the complex biofilm on the surface that also can be seen penetrating into the mouthguard porosities. (Original magnification: 2,000x.)

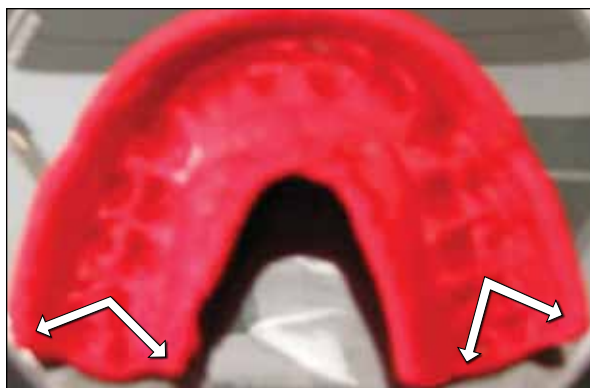


Fig. 3. The “boil and bite” mouthguard worn by a 13-year-old football player. Note the rough posterior edges (arrows).

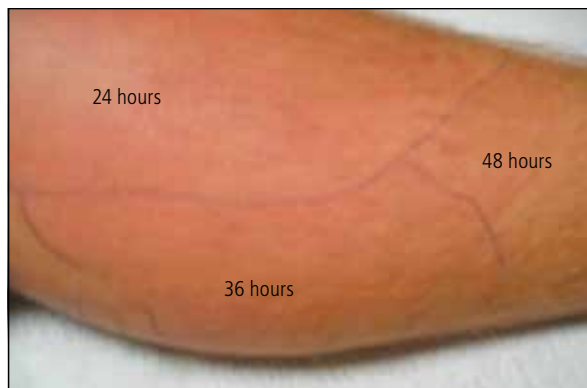


Fig. 4. The left leg of the patient in Figure 3. Note the substantial swelling over a period of 48 hours since the onset of symptoms (the numbers and black markings designate the swelling over that period of time).

been found in the mouthguards of football and ice hockey players, actual clinical cases have not been attributed to mouthguard wear. This article presents two cases of probable transmission of disease by contaminated mouthguards.

### Case report No. 1

A 13-year-old male football player began the season practicing twice a day, starting on August 20. Prior to his first practice, he form-fitted a “boil and bite” mouthguard (Fig. 3) and wore it until it was surren-

dered for testing on October 17 of that same year. The only significant dental finding was that the young man had full maxillary and mandibular orthodontic banding, which had been placed six months earlier.

On October 4, approximately six weeks after contact drills began, the young man’s lower left leg was injured when he was struck by an opponent’s helmet. There was no evidence of a visible bruise or laceration. His mother noted that he had ulcerations of his right buccal mucosa, the mucosa associated with

the left tuberosity, and the gingiva associated with teeth No. 5, 15, and 18, which she attributed to the mouthguard wear. He continued to practice and wear his mouthguard for another ten days. On October 15, his left leg became feverish, red, and swollen (Fig. 4).

Within 24 hours, the patient’s leg had swollen to approximately twice its normal size and he was barely able to walk. He was taken to the emergency room of the local hospital, where blood cultures were made and he was placed on IV antibiotics

(ampicillin). When he failed to respond to antibiotic treatment and the swelling continued to advance, he was transferred to a large metropolitan hospital. Upon his arrival at the emergency room, he was placed on IV vancomycin. When the swelling continued to increase and disseminate, the IV antibiotic was changed to nafcillin, a beta-lactam commonly used against MRSA. Over the next 24 hours, the swelling and cellulitis continued. At the same time, the lymph nodes in the left groin became tender and swollen. High levels of IV antibiotics were administered, alternating between vancomycin and nafcillin.

On October 20, the wound was incised and drained. The gram stain revealed Gram-positive cocci in short chains that were not further isolated or identified. However, a clinical diagnosis of MRSA was made solely on the basis of Gram stain and antibiotic response. The original blood cultures also were negative. Doppler examination of the left leg revealed no evidence that a deep vein thrombosis was an etiology for the cellulitis. The swelling slowly subsided over the next two weeks.

On October 17, the mouthguard (Fig. 3) was brought to the Infectious Disease Laboratory at Oklahoma State University Center for Health Sciences (OSU-CHS). Both the surfaces and depths of the mouthguard were cultured using standard aseptic techniques. While the cultures yielded isolated colonies of *S. saprophyticus* and *S. xylosus*, the predominant bacteria were Gram-positive *Stomatococcus mucilaginosus* and five isolates of *Bacillus* spp. In addition, six different isolates of the pink soil yeast *Rhodotorula* and five species of molds were identified. Antibiotic sensitivity tests were performed on the *Stomatococcus mucilaginosus*

and the microorganism was found to be susceptible to bacitracin, erythromycin, tetracycline, and (intermediately) to ciprofloxacin. It was resistant to oxacillin, penicillin G, gentamicin, and polymyxin B.

Considering the resistance patterns of the isolated microorganisms and the clinical response in this case, the most probable agent producing the cellulitis was *S. mucilaginosus*. The most feasible transmission route would have been from the mouthguard into the vascular system via the mucosal ulcerations. The noninvasive injury of the leg could have increased the blood flow in the region, allowing the vascular-disseminated *S. mucilaginosus* to invade the region of the injury and produce cellulitis.

### Case report No. 2

This 13-year-old boy began his football career in the first grade and routinely wore a custom-made mouthguard from that time to the present. By the end of his first season, he began to develop early symptoms of exercise-induced asthma (EIA). His pediatrician treated him with an oral bronchodilator inhaler. Over the next six years of football play, the subject's EIA symptoms varied but overall became progressively more intense. Midway through the 2006 football season, the subject had a severe asthma attack during practice which his bronchodilator could not control and he required additional medical attention. The subject's father brought the EIA to the attention of his dentist, who asked to have the mouthguard cultured. Within two days of the most recent asthma attack, the mouthguard was brought to the Infectious Disease Laboratory at OSU-CHS (Fig. 5).

The mouthguard was processed using standard aseptic techniques.



Fig. 5. The mouthguard of the patient in Figure 3. This mouthguard had been worn for approximately six weeks. Note the rough and jagged edges over the entire mouthguard surface.

The microorganisms on the surfaces and depths were cultured on blood agar, chocolate agar, and Sabouraud dextrose agar. The cultures revealed a variety of bacteria, including *Bacillus* spp., *Corynebacterium* spp., and *Listeria* spp. More importantly, six isolates of *Rhodotorula* spp. and five species of mold were isolated (Fig. 6).

Given the variety of yeasts and molds found in the mouthguard and the course of the subject's EIA, it is likely that the two are related etiologically. While exercising vigorously on the field, the subject presumably was aspirating yeasts and mold spores into his respiratory tract. There is a well-established connection between aspiration of yeasts and mold spores and the onset of asthma attacks in sensitized individuals. If the insult of the yeasts and mold spores was sufficient to cause EIA in this case, it probably would be refractory to the type of topical inhalation therapy that was attempted. Equally compelling is the finding that the subject also plays basketball but does not wear a mouthguard during

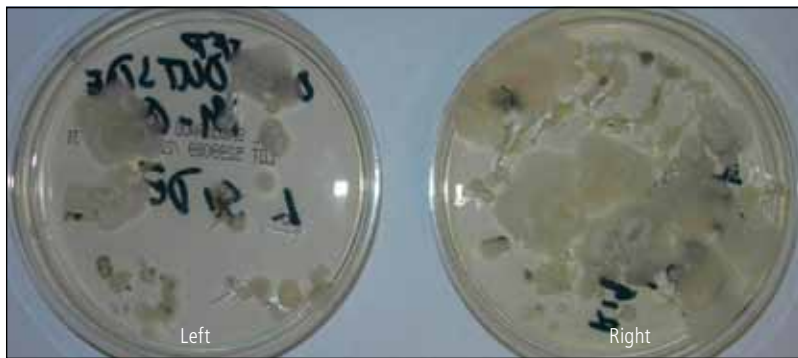


Fig. 6. Fungal cultures from the mouthguard in Figure 5. Note the quantity of yeasts and molds and the variety of colonial morphologies that represent different species.

practices or games and has had no evidence of EIA to date while playing this sport.

## Discussion

Two cases of serious disease were present in otherwise healthy young athletes. In both of these otherwise diverse clinical cases, the subjects played football and wore mouthguards that were contaminated with microorganisms that could be responsible for their illnesses.

In the first case, an unusual and seldom-seen opportunistic pathogen, *Stomatococcus mucilaginosus*, was cultured from the mouthguard and most likely was the agent cultured from the pus of the wound. Given the antibiotic resistance pattern of this subject's microorganisms and his clinical course of oral ulcerations, these findings would support an etiological association. Additional support for this assertion is found in several studies that point out the inconsistent antibiotic sensitivity patterns of *Stomatococcus mucilaginosus*. Other reports highlight that *Stomatococcus mucilaginosus* is rarely implicated as an etiological agent; however, it has been found in both local cellulitis (cervical necrotizing fasciitis) and blood-borne diseases

(such as endocarditis) that ultimately may lead to a cerebral mycotic aneurysm.<sup>23-25</sup> Other studies have reported that *Stomatococcus mucilaginosus* septicemia can be found in patients with chronic diseases and/or compromised immune systems.<sup>26-30</sup>

The second case report is associated with football but not basketball, suggesting that this athlete's condition does not strictly fulfill the criteria for classic EIA. Current theories of EIA etiology are related to changes in osmolarity of the bronchi and changes in the airway temperature.<sup>31</sup> However, most studies indicate that molds may function as allergens and therefore may be etiologic factors for EIA.<sup>32</sup> Allen noted in 2005 that bronchoconstriction had increased "among athletes from school children to Olympians to professionals."<sup>33</sup> A second article noted a similar increase in "asthma and bronchial hyperresponsiveness" that appeared to be more common among ice hockey players than in "floor-ball players" or the Swiss population in general.<sup>34</sup>

As a possible example of molds or yeasts causing EIA, the role of *Rhodotorula* spp. in the pathogenesis is intriguing, since this seldom-seen microorganism was isolated from

both of the mouthguards in this report. A previous study of college football players' mouthguards also isolated a large number of *Rhodotorula* spp.<sup>14</sup> It remains to be seen whether their presence in the mouthguards is mere coincidence or if they are active participants in a disease process such as EIA.

These findings raise interesting clinical conundrums. The characteristics of the microorganisms isolated from the mouthguards enable them to disseminate systemically and/or be aspirated into the respiratory tract. As a result, the immune system would be compromised and athletes would be more susceptible to diseases such as those demonstrated in these two cases. The spectrum of microorganisms found in mouthguards raises the question as to whether the risk from wearing mouthguards is worth the tooth protection they provide.

Finally, could these life- and/or health-threatening conditions be avoided by routine sanitization of the mouthguards or must the mouthguards be discarded on a regular basis? At the present time, there are no acceptable decontamination methods available. The major problem is that mouthguards, like dentures, are very porous. With use, microorganisms invade these porosities and thrive in the presence of food and water from their host athlete. Unfortunately, as with dentures, it is very difficult for sanitizing solutions to penetrate these pores. However, the biting actions athletes perform during mouthguard wear result in a systemic showering of microorganisms throughout the oral cavity, esophagus, and trachea. Based on the mouthguard studies to date, the authors recommend disposing of the mouthguards at least once a week. Another possibility is a single-use mouthguard. Regardless,



it is imperative that dentists advise patients who are athletes concerning the importance of proper mouthguard care.

### Summary

The two cases presented in this article strongly implicate disease transmission by mouthguard wear. While wearing a mouthguard to protect teeth is well-established and required in many sports, cases such as those presented in this article underscore the importance of proper mouthguard hygiene. Previous studies have shown that athletic mouthguards can harbor a wide range of opportunistic and frank pathogenic microorganisms. The rough and jagged nature of worn mouthguards allows for laceration of the oral tissues and for vascular dissemination of the microbes. The aspiration of the types of mold found in mouthguards could be an important etiologic factor in the rise of EIA seen in athletes. The authors recommend discarding mouthguards on at least a weekly basis. Because "boil and bite" mouthguards are so inexpensive, these might be considered as single-use mouthguards.

### Author information

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