DENTURE STOMATITIS: CAUSES, CURES AND PREVENTION

VICTORIA ELIZABETH HANNAH, LINDSAY O'DONNELL, DOUGLAS ROBERTSON, GORDON RAMAGE

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ABSTRACT

Removable dentures are worn by 20% of the UK population and two thirds of these individuals have denture stomatitis. Poor oral hygiene is commonplace among this group, as is smoking and xerostomia, which also contribute to the development of denture stomatitis. A complex polymicrobial biofilm is able to proliferate on the surface of denture materials and matures to form visible denture plaque. This denture plaque biofilm stimulates a local inflammatory process that is detectable clinically as erythema, and hyperplasia. Systemically, denture plaque represents a potential risk factor for systemic disease, in particular aspiration pneumonia. Respiratory pathogens have been detected in the denture plaque and overnight denture wear has been linked to an increased risk of aspiration pneumonia. There is a general lack of evidence on the adequate management of denture stomatitis and we present a protocol for use in the primary care setting.

KEY WORDS
Dentures, Stomatitis, Biofilms, Candidiasis, Pneumonia, Aspiration

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The presence of a denture in the mouth presents numerous challenges for both the patient and the clinician. By its very nature a denture is in close association with the oral mucosa, therefore irritation and inflammation is commonplace among denture wearers. Often patients find dentures difficult to tolerate, with ill-fitting dentures being known to impact negatively on quality of life.[1] Once finding a comfortable denture, individuals may wear the same denture for many years.[2] However, as the edentulous ridge continues to resorb, the result can be that a previously well-fitting denture becomes loose and traumatic.

Poor oral hygiene is commonplace among this patient group, with dentures providing a surface for microorganisms to adhere.[3] These adherent microorganisms very quickly form biofilms and visible denture plaque develops. The chronic exposure of the soft tissues underneath dentures to 'denture plaque' is associated with inflammatory changes in the soft tissues of the denture bearing area (see Figures 1 and 2). Denture stomatitis (DS) is the term widely used to describe this inflammatory change, and was first reported by GV Black as "sore mouth under plates" over 130 years ago. There are multiple patient factors that influence its onset and severity, including continuous denture wearing, denture cleanliness, denture trauma, salivary flow, denture base material, age of denture, smoking, dietary factors and pH of denture plaque though, microbial factors remain one of the most important.[4-7]

Denture stomatitis was classified by Newton in 1962 in terms of its severity ranging from mild, moderate to more severe soft tissue changes. Newton's type 1 presents as localised inflammation or pinpoint erythema (see Figure 3) and this is probably most frequently seen in patients with reasonable oral hygiene who do not remove their dentures at night. Newton's type 2 (see Figure 4) is a progression to more diffuse erythema involving part, or all, of the denture-bearing area. When the condition is long standing, patients may present with inflammatory papilliary hyperplasia (see Figure 5), usually on the hard palate and alveolar ridge (Newton's type 3). Other soft tissue pathology frequently accompanies DS, such as traumatic ulcers and soft tissue hyperplasia. Angular cheilitis can also co-exist with DS, particularly in immunocompromised or nutritionally deficient patients, and the denture is the primary reservoir for this mixed infection (see Figure 6). Frequently the patient's occlusal vertical dimension is incorrect, resulting in a reduced lower facial height and excessive creasing at the corners of the mouth where infection can develop. Patients are frequently unaware that they have DS as it is largely asymptomatic[8] in its milder forms, and so is usually an incidental finding at the patient's regular check-up. So why is it important?

Local and systemic implications of the dirty denture

We have an expanding elderly population, with 810 million people aged 60 or over worldwide. By 2050 this figure is expected to rise to at least two billion (22% of the entire global population). These ageing individuals generally experience a decline in oral health leading to tooth loss. This results in around 20% of the UK population wearing some form of removable denture, with a staggering 70% of UK adults over 75 years old wearing dentures.[1] More than two thirds of these individuals may suffer from DS.[8]

The oral and systemic link is not a new concept and has been well documented over the last 100 years, linking oral bacteria to rheumatoid arthritis, diabetes and cardiovascular disease. Dentures are in close proximity to the respiratory tract, therefore denture plaque biofilm represents a potential reservoir of opportunistic respiratory pathogens.[55] Aspiration of oropharyngeal contents is a common occurrence in healthy individuals, of whom approximately 45% aspirate material into the lungs during sleep.[9] This can potentially result in aspiration pneumonia (AP) in susceptible individuals. Risk factors for AP, such as dysphagia and chronic obstructive pulmonary disease, are more common in the elderly, and there is sufficient evidence available to support a relationship between dental plaque and pulmonary infection, particularly among hospitalised patients and the dependent elderly.[10-12]

Poor oral hygiene has already been linked to respiratory infection with common respiratory pathogens known to be present in both dental and denture plaque.[13] A recent study reported that patients who wear their denture overnight double their risk of developing pneumonia due to aspiration of opportunistic pathogens from the denture into their lungs.[14] A molecular analysis of the dentures of 131 subjects was performed by our research group and detected the putative respiratory pathogens *Staphylococcus aureus Haemophilus influenzae B, Pseudomonas aeruginosa, Streptococcus pneumoniae, Streptococcus pyogenes* and *Moraxella catarrhalis*

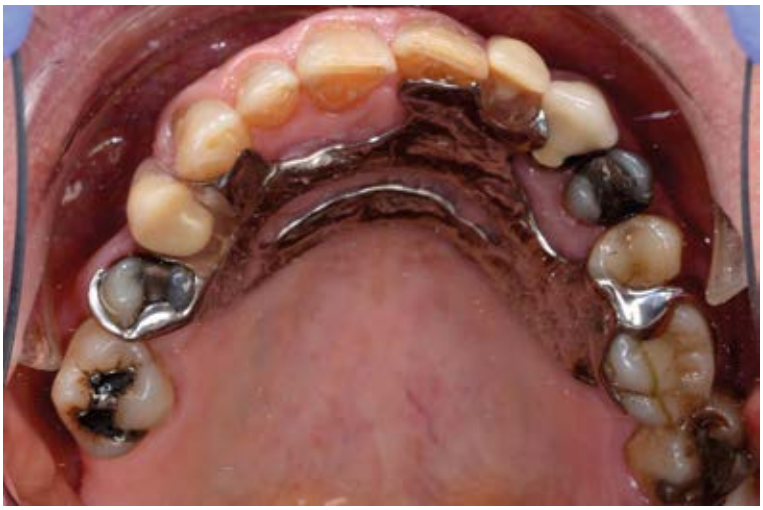


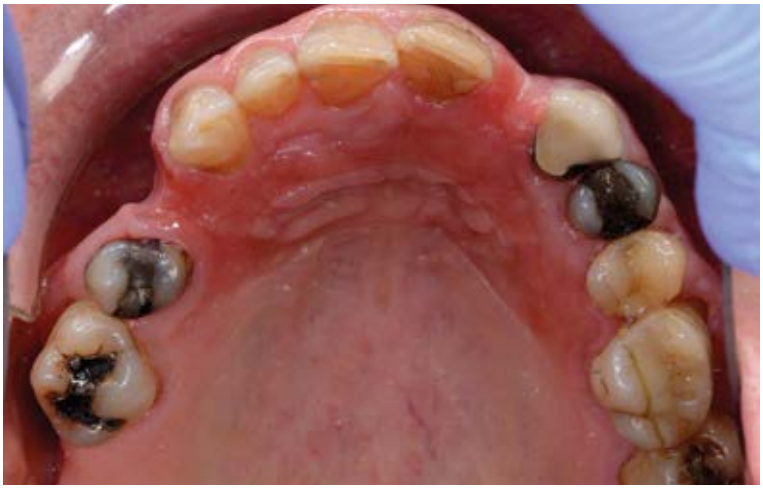
Figure 1: Upper cobalt chrome denture not removed at night

within denture plaque. Some dentures were colonised by up to three of these pathogens.[13] As pneumonia is the leading cause of death attributable to infection in patients aged 65 years and older, the presence of such species on our patient's dentures represents a potentially significant risk factor for the development of a life-threatening infection which costs the NHS in excess of £440 million annually.

The soft tissue changes observed clinically in DS sufferers are typically considered to be of yeast aetiology, with the literature disproportionately focused on *Candida* spp.[15-18] It could be inferred from this candidal bias that there is little else of microbial importance in the context of dentures, though in both healthy and diseased individuals it is apparent that denture plaque is more diverse than we assume.[19,20]

The denture surface is capable of carrying up to 10^{11} microbes per milligram.[21] Therefore, in terms of the wider systemic implications, dentures

Figure 2: Well demarcated denture stomatitis in denture bearing area



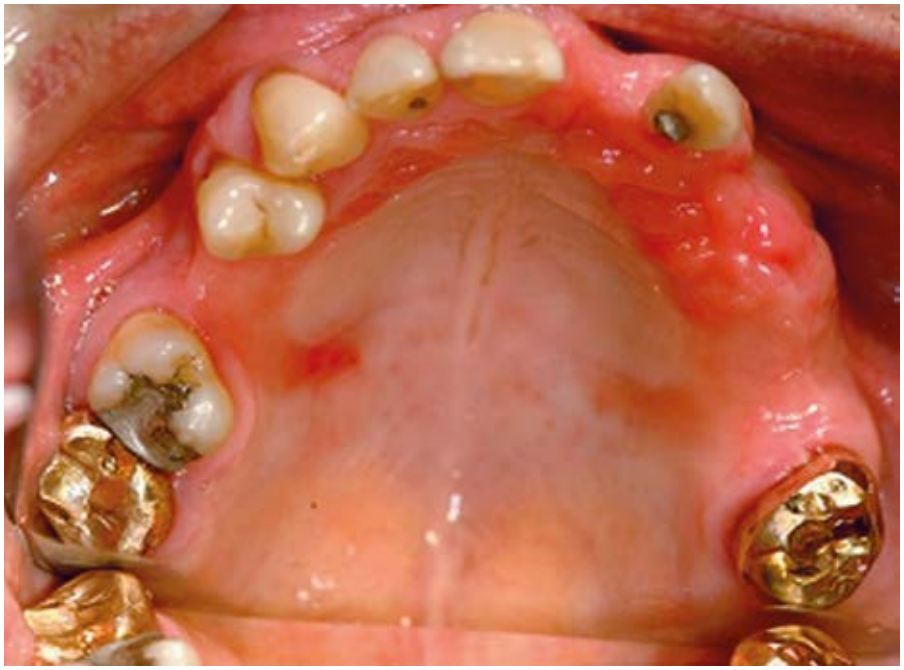


Figure 3: Newton's type 1 denture stomatitis

represent a significant reservoir for potentially pathogenic microbial species. The microbial composition of dental plaque has been well characterised and includes periodontal pathogens, including *Fusobacterium nucleatum, Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis)*[22,23] with caries-associated species being most prominent (*Streptococcus* and *Lactobacillus* species). Co-aggregation of these bacteria with *Candida albicans* hyphae,[24,25] enables these microorganisms to flourish as a biofilm within the topography of the denture surface with the collective active release of proteolytic and lipolytic enzymes inducing inflammation of the palatal surface,[26,27] ultimately leading to DS. Indeed, it has been reported that polymicrobial interactions lead to synergism and could potentially increase severity of DS.[28]

Is it just Candida?

DS is a multifactorial condition, and as with any disease the key to successful treatment is an understanding of the pathogenic microbial species involved. *C. albicans* was first identified a potential causative agent in DS in 1936 and is now the single most implicated species. *C. albicans* usually exists as a commensal in the oral cavity of 25-50% of the healthy population,[29] however under optimal conditions it can become pathogenic.[30] This switch to pathogenicity is related to its dimorphic capabilities, i.e. the ability to form hyphae and yeast interchangeably, a requisite of biofilm formation.[31] The hyphal form has been more commonly isolated in DS sufferers and is assumed to be the more invasive form of the organism, with an enhanced ability to adhere to and colonise the denture surface. There is some evidence to suggest that Candidal invasion may be secondary to soft tissue trauma and resultant inflammation.[32,33]

Host susceptibility also plays a significant role in the pathogenicity of candida species. As the population ages we are seeing patients with more complex medical conditions taking multiple drugs for their management. Those patients on

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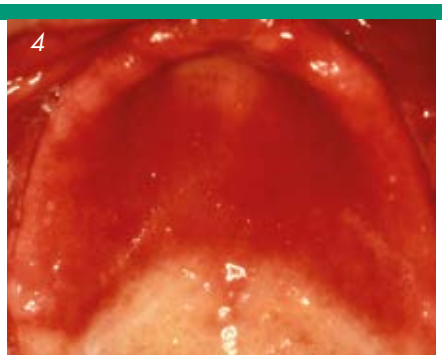

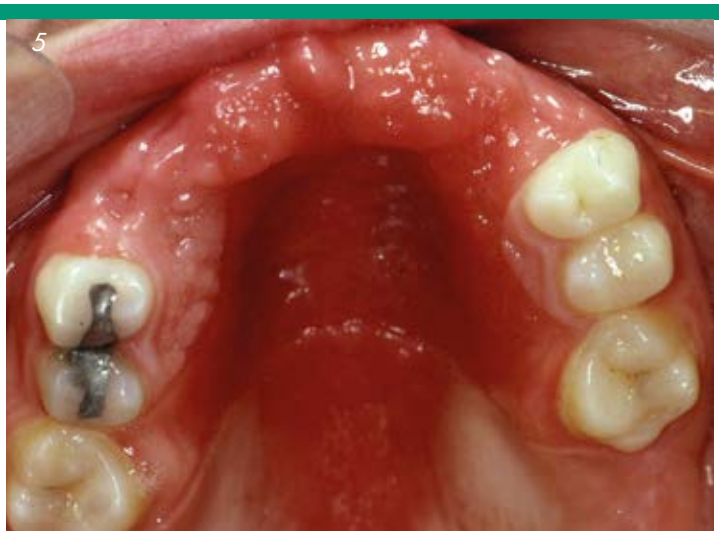

Figure 4: Newtons type 2 denture stomatitis

Figure 5: Newtons type 3 denture stomatitis

immunosuppressive therapies such as disease-modifying anti-rheumatic drugs, oral corticosteroids or chemotherapeutic drugs are particularly susceptible to candida infection and can develop widespread infection, such as osophageal candidiasis, if left untreated. Similarly, in patients with acquired immunocompromise due to HIV infection, susceptibility to candida infection is increased. Appropriate management of these patients involves treatment with antifungals and National Institute for Health and Care Excellence (NICE) guidance details suitable regimens and follow up. Integral to their management is the practice of good denture and oral hygiene.

At delivery, the surface of a denture appears to be smooth both visually and to touch. However, acrylic surfaces in particular are rough containing tiny cracks and fissures, which provide excellent habitats for microorganisms. Alveolar resorption is progressive following the loss of teeth, resulting in reduced retention and stability of dentures over time with resultant trauma to the underlying tissues. The acrylic surface also deteriorates during this time and a complex polymicrobial consortium of bacteria and yeasts set up residence within the cracks, fissures, and surface pores. It would be unwise to assume that *Candida albicans* was the only organism capable of existing in such an environment. In fact there has been a shift towards non-albicans species,[34-36] and *Candida glabrata* emerges as the second most prevalent species frequently isolated from denture acrylic[37] and patients with severe DS.[38] Despite *C. glabrata* lacking the biofilm formation capabilities of *C. albicans* it likely that *C. glabrata* uses *C albicans* as a structural scaffold to gain entry into the susceptible denture wearer. Moreover, it is only a matter of time before the emerging pan-resistant yeast *C. auris,* which has been shown to form biofilms, participates in the reservoir of infection on the denture surface.[39]

The microbiology of DS is complex, with over 30 bacterial phylotypes identified as unique to DS biofilms.[40] In the first study of its kind, our group has carried out a detailed analysis of the oral microbiome of 123 denture wearers using high throughput 16SrRNA gene sequencing technology. As expected, the most predominant species isolated was Candida (78% of dentures). Furthermore, *C.albicans* was co-isolated with *C.glabrata* in over a third of these DS cases, reinforcing the theory that *C. albicans* may act as a scaffold for non-biofilm forming species. The sequencing analysis revealed that the microbiome of partial denture wearers was significantly more diverse than complete denture wearers, both on the denture and mucosal surfaces. There were some notable changes between health and disease with the dentures of DS sufferers showing a more diverse microbiome with significantly higher proportions of *Prevotella spp* and *Veillonella spp* indicating a similarity with dental plaque biofilm. This initial study has lead us to believe that understanding the composition of the denture and mucosal microbiome is the key step to beginning to understand disease pathogenesis, and ultimately help improve treatments and identify novel targets for therapeutic and preventative strategies.

Prevention is better than the cure

The major aetiological factor associated with DS is poor denture hygiene. This can be compounded by a high carbohydrate diet, poor denture adaptation, smoking

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and reduced salivary flow. Approximately one half of denture wearers with poor fitting dentures are thought to experience localised Newton's type I inflammation.[38,41,42] This would suggest that to help prevent DS, attention should be paid to maximising the support, retention and stability of removable dentures. In xerostomic patients dentures are less retentive and more likely to traumatise dry friable mucosa,[43] so the ill-fitting denture in a patient with a dry mouth becomes more consequential in terms of mucosal inflammation and additionally quality of life.[44] Dry mouths favour the growth of acidogenic bacteria and *Candida spp.* This is likely to be related to the decrease in the surface roughness and surface free energy of acrylic resin when immersed in saliva.[37,45] The protective cleansing and immunological effects of saliva are lost in the xerostomic patient and candida adherence and colonisation is not impeded in the same way. Dry mouths require repeated lubrication to facilitate normal oral function. Patients must be educated with respect to the effects of dietary carbohydrate on the structural integrity of any remaining teeth, but also its effect of increasing candida growth and survival.[46] It may therefore be pertinent in the patient with recurrent DS to review dietary carbohydrate intake -and provide appropriate advice and alternatives. Several studies have shown that candidal oral carriage rate is significantly elevated in smokers when compared to non-smokers[47] so smoking cessation advice should be an integral factor in any DS treatment plan.

FIGURE 7

PROTOCOL FOR MANAGEMENT OF DENTURE STOMATITIS IN PRIMARY CARE

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Even in the non-xerostomic patient dentures are a barrier to the natural cleansing effects of saliva. Poorly cleaned dentures allow for the colonisation of yeasts and bacteria with a definite shift to a more pathogenic microbiome in those patients suffering from DS.[19] Sleeping with dentures in situ is still a common occurrence and plays a role in the aetiology of DS.[14,48,49] This practice does not allow time for natural salivary cleansing and means that the soft tissues of the denture-bearing area are never rested, with an increase susceptibility to local mucosal trauma. These factors can contribute to the development of an anaerobic environment between the denture and mucosa, favouring the growth of more pathogenic microbes, and has been related to increased risk of developing DS.[8]

Concluding thoughts

The future of treatment of DS may lie in the investigation of novel materials and coatings that provide a surface topography and chemistry that is more difficult for microbes to form biofilms on. Nanotechnology application to prosthodontic materials has been shown to be successful with composite resin, and its application to denture base materials continues to be investigated.[50] The application of coatings such as nano-polymers,[51] and nano-silica with diamond and platinum nano-particles[52] have been incorporated into denture acrylic[51] or used to modify the surface roughness to increase durapbilty and deter biofilm formation. The incorporation of antimicrobials into denture base materials and soft liners is not a new concept. However, the investigation of the antimicrobial application of natural compounds on denture associated biofilms is worth consideration. Curcumin (diferuloylmethane), the active ingredient of turmeric, is a polyphenol that demonstrates broad-spectrum antimicrobial properties. In a recently published study, curcumin was shown to adsorb to denture relevant substrates and to inhibit *C. albicans* adhesion, rather than actively kill or inhibit the microorganisms.[53] The application of such compounds as an alternative to commonly used antifungals is worth consideration given the growing trend of antimicrobial resistance. However, any new modality must be underpinned by an understanding of the microbiome in both health and disease if it is to be successful.

In terms of denture cleansing there is a generalised lack of evidence about the comparative effectiveness of different denture cleaning methods, and evidence is weak in support of soaking of dentures in effervescent tablets of enzymatic solutions.[54] Broad spectrum natural antimicrobial molecules have been investigated and may provide an alternative treatment for biofilm-related disease as development of antifungal resistance in Candidal species is commonplace.[55] At the highest level of evidence one systematic review concluded that there is no evidence that any denture cleaning method is more beneficial for the health of denture bearing areas or patients' satisfaction and preference when compared with another. However, a further systematic review by the same group investigating the treatment of denture stomatitis did suggest that Nystatin and disinfecting agents can reduce the inflammation associated with DS and the presence of *Candida* better than inactive treatment methods.[56] It has been suggested that disinfection methods, such as soaking dentures in chlorhexidine, and oral rinsing could have the same efficacy as antifungal medication, and a more conservative management approach would involve disinfection avoiding the side effects of common antifungals.[57] Antifungals should only be prescribed following culture and sensitivity analysis indicative of a susceptible candida species as recurrence rate and resistance is high in DS sufferers.[57]

So where does this leave us in terms of a denture cleansing management strategy for DS? The American College of Prosthodontists have published guidelines for the care and maintenance of dentures.[58] We present a modification of this protocol for management of denture stomatitis in primary care based upon best available evidence (see Figure 7).[54,56-58]

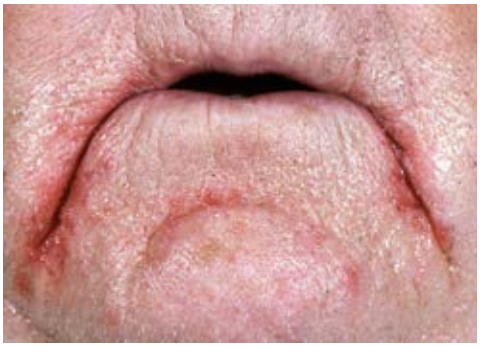

Figure 6: Severe angular cheilitis

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