

# Bridging the gap: an insight into dentistry

**EDUCATION** 

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No conflicts of interest to declare

Accepted for Publication: 26.04.17

#### **ABSTRACT**

#### Summary

Dental and medical care are closely interlinked since certain clinical oral manifestations can provide a useful insight into underlying systemic diseases. With recent research highlighting unique 'two-way' relationships between systemic and oral disease processes, it is paramount medical students are aware of these. Medical school curricula scarcely cover oral disease and oral examination; the purpose of this article is to provide a foundation for further learning. A clear focus has been made on four key aspects: the core dental team, oral lesions, dental disease and oral manifestations of systemic drugs.

# Relevance to medical students

Examination of the oral cavity forms an element of the wider physical exam. Hence, an appreciation for dentistry will place medical students in a more confident position knowing that they have an understanding of oral abnormalities. This will not only encourage improved diagnoses but discourage false referrals, making a positive impact on patient care and management.

#### Take home messages

Medicine and dentistry have a firm place in the primary health sector as patients share their health concerns with both their general medical practitioner and their general dental practitioner. Patient care can be greatly enhanced if a closer relationship between the two professions is fostered. This educational piece places a step in that direction by presenting to the reader an insight into the multifaceted speciality of dentistry.

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#### INTRODUCTION

Dental health can have a profound impact on the physical and emotional wellbeing of a patient. As such, dentistry is much more than the 'pearly smiles' portrayed in recent media. In many ways, the oral cavity can be seen as a "window on the body" (1), where oral changes can reflect underlying systemic illnesses, so a thorough oral examination may help medical practitioners confirm their diagnosis. (1) However, a recent study highlighted that 89% of medical students felt their training in the examination of the oral cavity was inadequate. (2) Furthermore, as a holistic approach to patient care is seen as the ideal, it makes sense that medical students have a working knowledge of dentistry. In their career, they are likely to encounter patients complaining of oral problems and having a basic knowledge of how to manage them will prove to be invaluable. This article aims to provide an initial insight into the world of dentistry.

#### THE DENTAL TEAM

As with medical care, dental care is delivered through a team approach (see figure 1). All members are equally important and must communicate effectively to ensure quality patient care. (3,4)

#### **ORAL LESIONS**

There is an array of lesions which present in the oral cavity, all differing in their clinical presentation. Whilst some may remain benign, some have the potential to turn malignant and can have a significant effect on a patient's life. (5) Figure 2 illustrates a range of lesions and their common clinical presentations.

White patch lesions can be the result of necrotic epithelium, debris or fungi collecting on the oral mucosal surface. (6) However, in some cases such as frictional keratosis, the white appearance can be attributed to an increase in keratinization. (6,7) This increase is brought about by recurrent mucosal trauma. (6,7) Trauma is not the sole reason as to why white patch lesions may develop. Further causes include neoplasia, immune related disease (e.g. lichen planus) and infection (e.g. candidosis). (6-9) Candida is ubiquitous as part of the normal oral microflora, however, its proliferation is generally the result of an underlying illness or antibiotic/steroid therapy. (7,10) Lichenoid reactions clinically resemble lichen planus (figure 3) and are brought about by systemic disease or a reaction to drugs and metals (such as amalgam). (8) Once the aggravating agent is removed, the reaction usually subsides. (8)

Erythematous lesions, like white patch lesions, also have many causes. Erosive lichen planus has a similar autoimmune aetiology to lichen planus however, here it presents as a painful lesion which is characterised by red patches. (8,11) Contact hypersensitivity

reactions are allergic reactions to materials used in dentistry such as amalgam, latex and anaesthetics. (12) The location of the erythematous patch usually corresponds to the site of exposure. (7) Erythematous candidosis is the term given when a candidal infection results in visible red areas on the oral mucosa in the absence of white plaque. (13) Taking a smear of the site can often indicate the presence of candida and the condition may itself be an indication of immunosuppression. (7,13)

Ulcerations of the oral mucosa are usually painful and they vary in severity and cause. Trauma to the oral mucosa results in a deep localised ulcer. (7) Conditions such as acute necrotizing ulcerative gingivitis (ANUG) which is thought to be caused by anaerobic bacteria and potentiated by factors such as stress and smoking, result in widespread ulceration affecting the gingivae. (14)

White patch lesions or erythematous lesions that cannot be rubbed off or characterised as a specific disease are termed leukoplakia or erythroplakia, respectively. (15) This subgroup of lesions has an increased malignant conversion rate and should be biopsied when detected. (6) Squamous cell carcinomas (figure 4) (SCC's) are the sixth most prevalent cancer worldwide and vary massively in their clinical presentation. (5) Heavy alcohol and tobacco consumption are key risk factors in the onset of SCCs and patients presenting with both are at a heightened risk. (16) Prognosis of SCCs are significantly improved with early diagnosis; although, their oftenpainless presentation may make this difficult. (15)

#### **INSIGHT INTO DENTAL DISEASE**

#### **Dental caries**

Although preventable, dental caries is a common progressive disease of the dental hard tissues, that can lead to pain and tooth loss if not managed appropriately. (17,18) Bacteria exist in symbiotic biofilms that form on the outermost surface of teeth. (17) When these bacteria metabolise dietary carbohydrates, acids are formed as a biproduct. (18) These acids can cause demineralisation and cavitation of the tooth surface. (17,18) Saliva acts as a natural buffer, which aids remineralisation. (19) Treatment options include working with the patient to improve oral hygiene, dietary advice, fluoride use and where necessary, restoring existing carious cavities. (19)

### **Apical Periodontitis**

Apical periodontitis is the acute or chronic inflammation of the periapical tissues which surround the apex of the tooth. (20) It usually follows untreated carious lesions which approach the pulp allowing the egression of bacteria periapically. (20) This is the main cause but other causes such as trauma can produce a similar situation. (20) Patients presenting with apical periodontitis may

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complain of a severe dull throbbing pain in the affected region, tenderness when biting and fever. (21) Root canal therapy is the treatment of choice and aims to resolve periapical inflammation through the removal of infected and necrotic pulpal tissue, but not all teeth can be saved and extractions may be required. (21)

#### Periodontal Disease (PD)

Although PD encompasses a range of disorders that affect the periodontium (a group of tissues which provide support to the teeth), the term commonly refers to gingivitis and periodontitis. (22) These are both inflammatory disorders brought about by a response to bacterial substrates from dental plaque. (22) Gingivitis is highly prevalent affecting many adults worldwide. (22) It is a reversible condition characterised by erythematous and swollen gingivae (gums) that bleed on brushing and is related entirely to plaque accumulation around the gingival tissues. (23) If allowed to progress it can result in a chronic irreversible condition where there is degradation of the periodontal tissues. (23) This is known as periodontitis and in severe stages can result in tooth loss. (22) Usually chronic periodontitis affects older patients, but in some individuals the disease is seen at a much younger age and is termed 'aggressive periodontitis'. (23)

Smoking, diabetes, medications affecting salivary flow, genetics, stress and immunocompromised states are all risk factors for periodontitis. (24) A bi-directional relationship between diabetes and periodontitis exists. (25) Studies show that hyperglycaemia can result in an increase in pro-inflammatory cytokines which can aggravate periodontitis and conversely, periodontal pathogens have been shown to increase insulin resistance. (26) It has been suggested that all patients with poorly controlled diabetes are referred for a periodontal examination. (24) Other bidirectional relationships with osteoporosis, respiratory and cardiovascular diseases also exist. (26)

The management of periodontal diseases involve improving the patient's oral hygiene and removal of supra- and sub-gingival calcified deposits. (27)

#### **ORAL MANIFESTATIONS OF SYSTEMIC DRUGS**

In an age where an increasing number of patients are being prescribed medications, knowledge of their side effects is crucial.

# Medication Related Osteonecrosis of the Jaw (MRONJ)

Bisphosphonates inhibit osteoclast function during periods of high bone turnover in disease states such as osteoporosis, Paget's disease and bone metastases. (28) MRONJ is a complication of bisphosphonate therapy (usually after dento-alveolar surgery) affecting normal bone turnover, characterised by areas of exposed, necrotic jaw bone. (29) Patients may present with pain, gingival ulceration and in advance stages, bone fracture. (28) Although emphasis is placed on preventing MRONJ, management involves frequent antimicrobial rinses and systemic antibiotics. (30) In severe cases, surgical intervention such as bone resection may be used. (30)

#### **Drug Induced Gingival Hyperplasia**

Three classes of drugs are associated with gingival hyperplasia (gingival overgrowth): anticonvulsants (e.g. Phenytoin), immunosuppressants (e.g. Cyclosporine) and calcium channel blockers (e.g. Nifedipine). (31) In a susceptible individual, gingival hyperplasia (figure 5) becomes clinically visible after 3 months from starting the medication. (32) Speech, aesthetics and mobility of teeth are all affected. (31) Minimising plaque levels, surgical intervention, tooth debridement and considering alternative medications are all treatment options. (33)

#### Xerostomia

Whilst it is widely acknowledged that many medications can lead to xerostomia (dry mouth), head and neck radiotherapy and disease states such as anxiety, depression, HIV and systemic disease such as diabetes and Sjögren's syndrome can also potentiate the condition. (34) Patients suffering from xerostomia will often experience eating and speech difficulties, taste disturbance, burning sensations and oral pain. (35) Furthermore, as the buffering effect of saliva is removed, the patient becomes more susceptible to dental caries. (36) Treatment for xerostomia aims to alleviate symptoms. Patients are advised to suck on sugar free sweets, use artificial saliva substitutes, drink more water and avoid dry foods. (36)

#### **CONCLUSION**

Although this article provides an insight into dentistry, it is by no means exhaustive. What has been covered should equip medical students with an appreciation of key areas in dentistry that they may encounter during their medical career. We hope that readers build upon this framework and develop their knowledge throughout their careers of lifelong learning. Medicine and dentistry go hand in hand and collaboration between members of both professions will result in the successful management of patients.

#### Acknowledgements:

We would like to thank Professor Alan Gilmour for his ongoing support, and Professor Michael Lewis for providing us with clinical photographs.

#### **FIGURES**

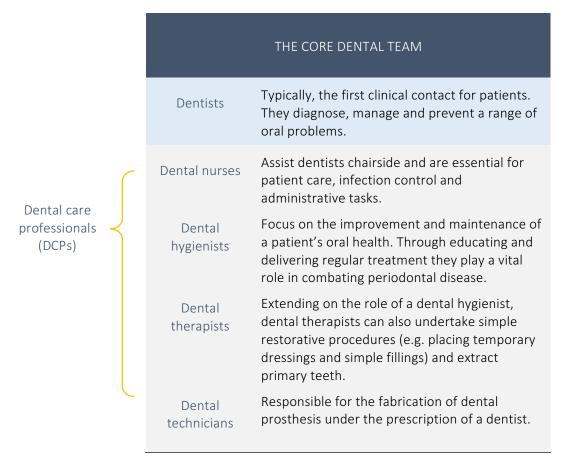


Figure 1 (table): Summary of the Core Dental Team. (4)

|             | DIFFERENTIAL DIAGNOSIS                         | CLINICAL PRESENTATIONS   |
|-------------|--|--|
| White patch | Candidosis                                     | <ul> <li>Creamy white or yellow patches</li> <li>May rub off (if at junction of the hard &amp; soft palates)/ may not (if at the corner of the mouth)</li> </ul> |
|             | Lichen planus (LP)                             | <ul><li>Symmetrical and bilateral</li><li>Often white striae on cheeks or tongue</li></ul>   |
|             | Lichenoid reactions                            | <ul><li>Appearance resembles LP</li><li>Presents asymmetrically</li></ul>  |
|             | Frictional keratosis                           | <ul> <li>White lesion which may appear thick and corrugated</li> <li>Often presenting on the cheeks/lateral margins of the tongue</li> </ul>                     |
|             | Leukoplakia                                    | <ul><li>White patch of unknown cause</li><li>Cannot be wiped off</li></ul>   |
|             | Squamous cell carcinoma<br>(SCC)               | <ul><li>Varying presentations</li><li>Commonly on the tongue/retromolar region/ floor of the mouth</li></ul>   |
| Erythema    | Erosive lichen planus                          | <ul><li>Red patch lesion</li><li>With/without white striae</li></ul>   |
|             | Erythematous candidosis                        | <ul><li>Painful red areas</li><li>Common on the palatal mucosa</li></ul>   |
|             | Contact hypersensitivity reactions             | <ul><li>Red patches</li><li>Potentially presenting with vesiculation</li></ul>   |
|             | Erythroplakia                                  | <ul><li>Red patch of unknown cause</li><li>Cannot be wiped off</li></ul>   |
|             | Squamous cell carcinoma (SCC)                  | <ul><li>Varying presentations</li><li>Commonly on the tongue/retromolar region/floor of the mouth</li></ul>  |
| Ulceration  | Traumatic ulceration                           | Single localized ulcer   |
|             | Acute necrotizing ulcerative gingivitis (ANUG) | <ul><li>Painful ulceration at gingival margin</li><li>With halitosis</li></ul>   |
|             | Squamous cell carcinoma (SCC)                  | <ul> <li>Varying presentations</li> <li>Commonly on the tongue/retromolar region/floor of the mouth</li> </ul>   |

Figure 2 (table): Summary of Specific Oral Lesions. (5-16)



Figure 3 (photograph): Lichen Planus – photograph courtesy of Professor M A O Lewis, Cardiff University



Figure 4 (photograph):
Squamous Cell Carcinoma –
photograph courtesy of
Professor M A O Lewis, Cardiff



Figure 5 (photograph): Gingival Hyperplasia – photograph courtesy of Professor M A O Lewis, Cardiff University

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#### Journal DOI

10.18573/issn.2514-3174

# Issue DOI

10.18573/n.2017.10170

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