

# Managing patients with rheumatoid arthritis

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**R**heumatoid arthritis (RA) is a common chronic inflammatory autoimmune disorder which significantly impacts patients' lives and can lead to permanent disability. Inflammation in RA not only affects joints; it can affect organs including the heart and lungs. Early diagnosis, initiation of intensive drug therapy, and a multidisciplinary care approach have vastly improved the long-term prognosis for those living with the condition. However, RA patients often present with co-morbidities which add to the complexity of clinical management. Orofacial conditions associated with RA which dental professionals need to be aware of include periodontal disease, temporomandibular dysfunction and salivary gland dysfunction. In this article, we provide information on RA, oral health in RA and guidance on how

best to manage patients with RA in general dental practice.

## Background

Rheumatoid arthritis (RA) is a common chronic inflammatory autoimmune condition which affects an estimated 400,000 UK adults.<sup>1</sup> Onset is commonly between 40–50 years old, though can occur at any age, with women three times more likely affected than men.<sup>1</sup> RA is a common cause of disability; work disability increases with age and disease duration.<sup>2</sup> RA results in increased healthcare costs and use of social security provision, as well as significantly reducing a patient's quality of life.<sup>3,4</sup>

## Pathophysiology

RA is characterised by the production of rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPAs) against autoantigens commonly expressed within and outside of synovial joints.<sup>5</sup> RF is produced by B cells in the synovial membrane and is associated with more aggressive joint destruction.<sup>6</sup> Traditionally, RF was the classic autoantibody in RA; however, ACPAs are now seen as being of increased importance as they are more specific and sensitive for RA diagnosis and predict a poorer disease course with progressive joint damage.<sup>7</sup> RF, ACPA or both are present in 50–80% of people with RA.<sup>7</sup>

RA has several distinct disease subsets with several inflammatory cascades all resulting

in persistent synovial inflammation with damage to articular cartilage and underlying bone.<sup>7</sup> Key inflammatory cytokines in its pathogenesis are tumour necrosis factor alpha (TNF- $\alpha$ ) and interleukins 1 and 6 (IL-1, IL-6), which result in the production of proteolytic enzymes and activation of osteoclasts.<sup>6–8</sup>

## Aetiology

It is thought RA occurs in response to environmental triggers in genetically susceptible individuals.<sup>5</sup> The most recognised environmental trigger is smoking, which increases levels of the peptidyl arginine deiminase (PAD) enzyme responsible for protein citrullination (the conversion of arginine to citrulline).<sup>9</sup> A recent study identified a clear dose-response relationship between smoking and the risk of developing RA.<sup>10</sup> The periodontal pathogen *Porphyromonas gingivalis* has been implicated in the aetiology of RA as has, most recently, the gut microbiome.<sup>11,12</sup> Other suggested environmental risk factors include region of birth, birthweight, breastfeeding and socioeconomic status.<sup>13</sup> Genetic factors account for 50% of the risk of RA development, with more than 100 loci for genetic susceptibility identified to date.<sup>14,15</sup> ACPA presence is associated with alleles containing a shared epitope (common protein-binding motif) in the HLA-DRB1 locus, and can be detected up to 15 years before RA onset.<sup>16,17</sup>

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### Signs and symptoms

Common clinical features of RA include a symmetric polyarthritis with joint swelling (particularly in the hands and feet).<sup>18</sup> Persistent joint inflammation (synovitis) results in bone and cartilage destruction which can ultimately lead to deformity, chronic pain and a loss of function.<sup>9</sup> Patients can experience stiffness upon waking or after prolonged periods of rest.<sup>18,19</sup> Systemic inflammation in RA can affect the brain (fatigue, reduced cognitive function and cerebrovascular events), liver (elevated acute-phase response and anaemia), heart (myocardial infarction and cardiac failure), lungs (inflammatory and fibrotic diseases), exocrine glands (secondary Sjögren's syndrome), bone (osteoporosis) and muscles (sarcopenia).<sup>20</sup> In severe RA, subcutaneous nodules and other extra-articular manifestations (such as vasculitis) can develop.<sup>18</sup>

### Diagnosis

In 2010, the European League Against Rheumatism (EULAR) and the American College of Rheumatology (ACR) jointly released new classification criteria for RA to assist rheumatologists with the diagnosis of newly-presenting patients.<sup>21</sup> This assesses joint involvement, autoantibody status, acute-phase reactants and symptom duration.<sup>7</sup> Early diagnosis and initiation of intensive treatment prevents joint damage, and greatly improves functional outcome and morbidity for patients.<sup>18</sup>

### Clinical assessment

Disease activity is often assessed using combined indices such as the Disease Activity Score, based on the 28 joint count (DAS-28) which assesses joints in the hands, arms and knees for swelling and tenderness; measures the erythrocyte sedimentation rate (an inflammatory marker), and the patient's global assessment (a visual analogue scale-based score the patient gives for how RA is affecting them overall at that time).<sup>6</sup> The Simplified Disease Activity index (SDAI) or the Clinical Disease Activity index (CDAI) are alternatives.<sup>6</sup> X-rays, ultrasound and magnetic resonance imaging are all used to assess structural changes in the joints.<sup>6</sup>

### Medical management

The aim of drug therapy is to reduce symptoms and suppress inflammation, thereby limiting joint damage and disability.<sup>22</sup> Currently, it is recommended to follow a 'treat-to-target' strategy which involves initiating intensive drug therapy

immediately after diagnosis and escalating this, guided by disease activity assessment, until clinical remission or low disease activity is achieved.<sup>20,22</sup> Disease-modifying anti-rheumatic drugs (DMARDs) are the primary treatment used as they reduce pain and swelling of the joints, lower levels of acute-phase inflammatory markers, limit progressive joint damage and improve function.<sup>7</sup> The first DMARD usually prescribed is methotrexate (sulfasalazine or leflunomide can be given if methotrexate is contraindicated).<sup>7</sup> DMARDs can be used in combination to be more effective.<sup>23</sup>

serious infection (for example, tuberculosis) over conventional DMARD use, with this risk at its highest during the first six months of therapy.<sup>27,28</sup> Other infection risks are bacterial (for example, sepsis, abscesses and cellulitis), fungal (for example, candidosis) and viral (for example, shingles).<sup>7,29</sup> Although there has been speculation about an increased risk of developing cancer with biologics use, a recent systematic review has shown this to be little or none.<sup>30</sup> In future, it may be possible to taper biologic DMARDs in select patients whose RA is in remission.<sup>31</sup>

Improved management of RA over the past

## 'SYSTEMIC INFLAMMATION IN RHEUMATOID

## ARTHRITIS CAN AFFECT THE BRAIN,

## LIVER, HEART, LUNGS, EXOCRINE

## GLANDS, BONE AND MUSCLES'

Conventional DMARDs modify the whole immune system, whereas biologic DMARDs target specific components within the inflammatory pathway.<sup>6</sup> Biologic DMARDs are given to patients with persistently active disease, and are highly effective.<sup>22</sup> They are usually prescribed for use in combination with methotrexate, as it is thought that this both reduces antibody formation to the biologic agent and increases its efficacy.<sup>7</sup> Smoking reduces the efficacy of conventional DMARDs and smokers are more likely to fail on biologic therapies.<sup>24</sup>

In RA, the pain response appears to be heightened; analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) can be used for additional symptom control.<sup>7,25</sup> Due to unfavourable side-effects, corticosteroid use is restricted to bridging therapy during acute flares of symptoms ('flare-ups') whilst awaiting DMARDs to reach full efficacy.<sup>7</sup> Intra-articular injections are highly effective for disease suppression in individual active joints.<sup>7</sup> New biologic DMARDs are currently in development, whilst biosimilar drugs and novel small molecule agents have come onto the market.<sup>6,22,26</sup> The long-term effect of these drugs in clinical practice is yet to be determined. See Table 1 for a list of the most common drugs currently used to manage RA.

Having RA increases the risk of infections and this is raised further with the use of biologics.<sup>6</sup> Biologic DMARDs have approximately a 30% increased chance of

two decades has resulted in a decline in its severity with less inflammatory biomarkers, extra-articular manifestations, hospital admissions and joint replacements.<sup>7</sup> As better therapies have resulted in the reduction or absence of deformities, RA patients can appear quite 'normal'.<sup>32</sup> For effective RA management, adjunct non-pharmacologic interventions are also advised (such as patient education, physiotherapy, occupational therapy, foot care and psychological support) which are delivered by a multidisciplinary team of healthcare professionals.<sup>33</sup>

### Co-morbidities

Co-morbid conditions can be coincidental (with some already present at RA diagnosis), reflective of RA or its treatment.<sup>34</sup> RA patients have excess mortality from cardiovascular disease (CVD), but it remains unclear whether cardiovascular events are caused by inflammation associated with RA rather than traditional cardiac risk factors (such as hypertension, dyslipidemia and cigarette smoking).<sup>35,36</sup> DMARDs are associated with a decreased risk of all cardiovascular events but this is increased with use of NSAIDs and corticosteroids.<sup>37</sup>

After CVD, the second most common cause of mortality in RA is from cancer, particularly lymphoma and lung cancer.<sup>38,39</sup> Patients with RA are more likely to develop skin cancer (non-melanoma and possibly melanoma) than the general population, and this risk

may be increased by DMARD use.<sup>40–42</sup> RA patients have an increased risk of osteoporosis and resultant bone fractures linked to age, disease duration and steroid therapy.<sup>38</sup> Other co-morbidities include anaemia, depression, fibromyalgia, interstitial lung disease, Sjögren’s syndrome, periodontal disease, diabetes mellitus and obesity.<sup>7,34,38,39,43–46</sup>

It was previously reported that RA patients die prematurely from one or more comorbid diseases.<sup>47</sup> However, a recent study found that people who had RA onset after 2000 no longer have an increased risk of mortality compared to the general population.<sup>48</sup> This is likely due to improved treatments and tighter disease control over the last two decades. Overall, comorbidities increase disability levels, reduce a patient’s quality of life, make patient management more complex and increase the economic burden of disease.<sup>34,38</sup>

**Oral health**

Oral health complications due to RA and its treatments can cause additional problems for patients. A recent study found that approximately 30% of RA patients were taking additional analgesics specifically for oral pain.<sup>49</sup> Due to their immunosuppressive effects, RA medications can promote periodontitis, candidosis and oral ulceration aided by a lack of saliva.<sup>50</sup> The three main oral conditions associated with RA are discussed below.

**Periodontal disease**

Periodontal disease (PD) is a chronic inflammatory condition which leads to destruction of the periodontal ligament and alveolar bone, and can result in tooth loss.<sup>51</sup> PD is caused by the presence of pathogenic gram-negative anaerobic bacteria within the biofilm attached to the sub-gingival tooth surface.<sup>9</sup> *Porphyromonas gingivalis* (Pg) is the main pathogen in PD.<sup>9</sup> Its virulence combined with an intense host immune response is thought to contribute to the severity of the disease.<sup>9</sup>

People with RA are almost twice as likely to have PD than those without.<sup>52</sup> RA patients with severe PD have significantly higher DAS-28 scores than those with moderate or no periodontitis, and PD is associated with increased radiographic joint damage.<sup>53,54</sup> These data strongly suggest an association between RA and PD/tooth loss. This association is independent of common risk factors such as smoking, alcohol intake, socioeconomic background and poor oral hygiene.<sup>55</sup>

RA and PD are both chronic inflammatory diseases. Both conditions feature excessive

**Table 1 Drugs commonly used in RA management<sup>6,22</sup>**

Drug class	Generic names
Analgesics	Paracetamol
	Co-codamol
	Co-dydramol
	Tramadol
NSAIDs	Ibuprofen
	Naproxen
	Diclofenac
Corticosteroids	Prednisolone
	Methylprednisolone
Conventional DMARDs	Methotrexate
	Leflunomide
	Sulfasalazine
	Hydroxychloroquine
Biologic DMARDs	Adalimumab
	Etanercept
	Infliximab
	Certolizumab pegol
	Golimumab
	Rituximab – B-cell depletor
	Abatacept – T-cell costimulation inhibitor
	Tocilizumab – IL-6 inhibitor
Small molecule agents	Tofacitinib – JAK inhibitor
	[More to be licensed]

NSAIDs = Non-steroidal anti-inflammatory drugs; DMARDs = Disease-modifying anti-rheumatic drugs; TNF = Tumour necrosis factor; IL-6 = Interleukin-6; JAK = Janus kinase.

destruction of collagen-rich tissues: in RA these are bone, cartilage and other periarticular tissues; in PD these are alveolar bone, periodontal ligament and gingiva.<sup>56</sup> Alveolar bone loss in PD results from the activation of osteoclasts and is very similar to bone erosion in RA, which is caused by cytokine-driven osteoclast activation.<sup>9</sup>

PD may be involved in the initiation and/or maintenance of systemic inflammation in RA.<sup>9</sup> The level of Pg antibodies has been found to positively correlate with levels of ACPA in circulation in RA.<sup>57</sup> Pg is the only bacterium known to express a PAD enzyme, which can cause the citrullination of bacterial and host proteins.<sup>58</sup> This is thought to cause the body to produce ACPA, which drives the autoimmune response in RA.<sup>58</sup> PAD enzymes, citrullinated proteins and ACPA have all been found in inflamed gingiva.<sup>59,60</sup> Antibodies to the periodontal pathogens Pg and *Prevotella intermedia* have been found in the serum

and synovial fluid of patients whose RA is active.<sup>61,62</sup>

Effective control of PD for RA patients is important to reduce both local and systemic inflammation, and the likelihood of bacteraemia.<sup>63</sup> Persistent periodontitis can also reduce the effectiveness of TNF inhibitors.<sup>64</sup> Short-term clinical trials have demonstrated that non-surgical periodontal therapy in RA patients with PD can decrease RA disease activity and systemic inflammation.<sup>65–67</sup> Reduction in disease activity may be due to less inflammatory products, bacteria and endotoxins in the bloodstream after periodontal treatment, thereby reducing the exposure of joints to these products.<sup>68,69</sup> Longer-term clinical trials are currently in progress to find out whether non-surgical periodontal treatment can lead to an improvement in clinical outcomes and quality of life for patients with active RA.<sup>70</sup> A recent systematic review also highlighted

**Table 2 Oral signs and symptoms of Sjögren's Syndrome**<sup>75,88,90,91</sup>

Signs	Symptoms
Enlarged parotid glands	Soreness
Absence of saliva pooling in floor of mouth	Burning
Dry/cracked oral mucosa and lips	Loss of/altered taste sensation
Mouth sores	Difficulty with eating
Increased caries incidence (especially cervical)	Difficulty with swallowing
Increased dental erosion	Difficulty with speaking
Erythematous cobblestoned/fissured tongue	Pain from denture-induced irritation
Atrophy of filiform papillae	
Coated tongue ('black hairy tongue')	
Candidosis	
Halitosis	
Difficulty with denture retention	

**Table 3 Suggestions to make dental visits more comfortable for RA patients**<sup>93,94</sup>

<b>Before treatment</b>	Organise appointments at times more suitable for the patient's condition; for example, if they experience morning stiffness, schedule visits for the afternoon.
	Make shorter, more frequent appointments rather than lengthy visits where patients can experience stiffness in the dental chair.
	Book a treatment room with step-free access.
<b>During treatment</b>	Adjust the dental chair and headrest to a comfortable position, as RA patients can get considerable neck pain and stiffness during treatment.
	Offer the patient a small pillow or allow them to bring their own.
	Allow the patient to rest and move their jaw periodically during treatment to prevent pain, fatigue and stiffness from keeping their jaw open for prolonged periods.
	Reassure the patient that they can ask to take a break at any time.
	Ask the patient if they require any other adjustments.

the importance of smoking cessation, which results in improved outcomes for non-surgical periodontal therapy.<sup>71</sup>

### Temporomandibular dysfunction

The temporomandibular joint (TMJ) is used up to 2,000 times a day for chewing and speaking, making it one of the most frequently used synovial joints in the body.<sup>72</sup> People with RA have a higher frequency and greater severity of temporomandibular dysfunction (TMD) than the normal population.<sup>73</sup> The estimated prevalence of TMJ symptoms in adults with RA is between

5–86% (depending on diagnostic criteria, assessment methods and the population studied) with clinical involvement of the TMJ seen in about 50% of cases.<sup>73,74</sup> In a 2013 survey of RA outpatients at a tertiary centre, 45.8% had problems with chewing (with 40.3% having to adjust their diet accordingly), 30.6% felt discomfort when eating and 36.1% took medication to relieve oral pain.<sup>49</sup>

RA patients with TMD may present with pain, difficulty with opening the mouth, 'locking' of the jaw, tenderness of the TMJ/masticatory muscles, and joint sounds.<sup>73,75</sup> The most frequent joint sound is clicking,

followed by crepitus (which indicates TMJ degeneration but may be seen less often due to improved RA medication).<sup>76</sup> It is thought that pain in TMD is associated with RA disease activity, and impairment in the range of motion and function of the TMJ are more likely due to degeneration of the joint.<sup>73</sup> Patients may also report associated symptoms such as ear pain/stuffiness, tinnitus, dizziness, headache and neck pain.<sup>77</sup> *It is important to note that the TMJ may already be affected by RA in patients who do not yet report TMD symptoms.*<sup>78</sup>

Clinical signs of TMJ involvement include swelling, reduced range of motion and/or deviation of the mandible to the affected side.<sup>75</sup> Imaging shows condylar resorption with a resultant shortening of the mandibular ramus-condyle unit and possibly a reduced joint space.<sup>77</sup> Cone-beam computed tomography (CBCT) imaging is best for showing the extent of condylar damage from RA, particularly in the early stages, and involves a lower radiation dose than conventional CT scans.<sup>79</sup> There is a positive correlation between the duration and severity of RA, and the degree of TMJ involvement.<sup>78</sup> Ankylosis of the TMJ is uncommon and occurs late in the disease course.<sup>74</sup> If ankylosis or collapse of the TMJ occurs, joint replacement may become necessary.<sup>74</sup> This has been shown to have good long-term outcomes for patients with inflammatory arthritis.<sup>80</sup> *TMD management in RA needs to involve the patient's rheumatologist and an oral and maxillofacial surgeon with an interest in TMJ diseases.*<sup>74</sup>

Juvenile idiopathic arthritis (JIA), also known as juvenile rheumatoid arthritis, affects around 12,000 children in the UK.<sup>81</sup> The reported prevalence of TMJ arthritis in JIA ranges from 17 to 87%.<sup>82</sup> Restricted mouth opening is the most frequent clinical finding (28% of patients) followed by masticatory muscle tenderness, deviation of the mandible on opening, TMJ tenderness and joint sounds.<sup>82</sup> TMJ arthritis in children can cause a disturbance of mandibular growth and evident alterations in craniofacial morphology and occlusion; features typically seen include an increased profile convexity, a steeper mandibular plane angle, mandibular micrognathia and retrognathia.<sup>83</sup>

Inflammation occurs during the active phase of JIA, which ultimately causes resorption of the condyles.<sup>82</sup> Damage to the condyles may be present early on in JIA and progress, even when clinical symptoms and signs are absent.<sup>82</sup> The current gold standard method of imaging in JIA to detect early arthritic changes in the TMJ is magnetic

resonance imaging (MRI) with contrast.<sup>84</sup> Patients with JIA should have regular imaging of the TMJ and evaluation by an orthodontist, even in the absence of TMD signs and symptoms.<sup>82</sup>

Lastly, it is important to note that some patients with RA/JIA will have TMD that is unrelated to their inflammatory arthritis.

**Salivary gland dysfunction**

Sjögren’s syndrome (SS) is a chronic autoimmune condition that is characterised by the sicca symptoms xerophthalmia and xerostomia, caused by inflammation leading to dysfunction of the lacrimal and salivary glands.<sup>85</sup> It can occur alone (primary form) or secondary to other systemic autoimmune diseases such as RA.<sup>86</sup> The estimated prevalence of sicca symptoms in RA patients ranges between 30–50%.<sup>86</sup>

RA patients with SS have reduced salivary flow and altered saliva composition (due to destruction and dysfunction of the salivary glands).<sup>87</sup> This reduces the buffering and antimicrobial properties of saliva causing an increased likelihood of caries.<sup>88</sup> The subjective experience of xerostomia in SS is not dependent on the total quantity of saliva (salivary flow) but rather the quantity and quality of specific components within it (called mucins) which affect its ability to retain water.<sup>87</sup> Up to 70% of salivary mucins are produced by the minor salivary glands and overall they produce 10% of saliva.<sup>89</sup> Therefore, currently available treatments aimed solely at increasing salivary flow may not be sufficient to provide relief for SS patients.

Xerostomia can affect the oral cavity in many ways (see Table 2). Besides SS, a dry mouth can also be caused by medications taken by RA patients (see Table 1). It is important to note that xerostomia may be reported by patients before obvious signs of hyposalivation are visible in the mouth.<sup>87</sup> *If a patient presents with a dry mouth and also complains of dry eyes, it is worth writing to their general medical practitioner (GP), or rheumatologist if they have a known rheumatic disorder, for further investigation.*

**Dental management**

*General considerations*

Most RA patients can be successfully managed at the dental practice with some minor adjustments – see Table 3 for suggestions. Chronic inflammation of the cervical spine in RA can result in neck instability, which can cause neurological symptoms and in rare cases be fatal.<sup>92</sup> *It is therefore important that a patient’s head and neck are well supported*

**Table 4 Summary of suggested management for RA-associated dental problems**<sup>74,75,77,88,90,91,93–95,97–100</sup>

Dental problem	Management
Periodontal disease	More frequent dental/hygiene visits
	Regular scaling and root planing (no adjuncts necessary)
	Oral hygiene instruction – recommend electric toothbrushes and interdental cleaning aids with wider handles
	Smoking cessation advice and support
	Refer to a periodontist if necessary
Temporomandibular dysfunction	Jaw rest
	Warm compress application
	Physiotherapy
	Soft food diet
	Short-term NSAID use (topical or systemic)
	Occlusal splint (soft or hard) wear at night time
	Biobehavioural therapy
	Elimination of unhelpful habits for example, nail biting, wide yawning
	Discuss with patient’s rheumatologist/GP if TMJ arthritis suspected
	Refer to oral & maxillofacial surgery and orthodontics (for children) if necessary
Salivary gland dysfunction	More frequent dental visits
	Medication review
	Advise to keep hydrated with regular sipping of water
	Smoking cessation advice
	Chew sugar-free gum or lozenges regularly (if no TMJ problems)
	Oral hygiene instruction
	Pit and fissure seal teeth
	Fluoride varnish, prescription-strength toothpaste or mouthwash
	Use of non-fluoride remineralising agents for example, calcium phosphate rinse
	Chlorhexidine varnish, gel or mouth rinse
	Advise to reduce sugar/acid intake and frequency
	Salivary replacement (gels, mouth rinses, toothpastes, lozenges)
	Advise patient to use a humidifier, particularly when sleeping
	Discuss with patient’s rheumatologist/GP
	Prescribe salivary stimulants for example, pilocarpine
	Refer to oral medicine if necessary
Refer to GP/rheumatologist if an undiagnosed underlying rheumatic disease is suspected	
Oral candidosis/ angular cheilitis	Prescribe topical or systemic antifungals
	Discourage denture wear at night
	Encourage good denture hygiene
Oral ulceration	Check patient is taking medication (especially methotrexate) at the prescribed dose and interval
	Prescribe benzydamine mouthwash/oromucosal spray
	Urgent referral to oral medicine if ulcers are longstanding (>3 weeks) or suspicious

NSAID = Non-steroidal anti-inflammatory drug; GP = General medical practitioner; TMJ = Temporomandibular joint.

during dental treatment. Suspected cervical instability should also be discussed with the patient's rheumatologist.

Due to pain, impaired hand function and fatigue, RA patients may find it difficult and lack the motivation to follow a good oral hygiene regime (leading to further unfavourable outcomes).<sup>54,55,95,96</sup> Good oral hygiene is the cornerstone for dental management of these patients, and aids should be recommended to making brushing and interdental cleaning easier for this population with poor grip and dexterity. Resources on suitable aids and adaptations are available.<sup>93,95</sup> See Table 4 for a summary of management of common RA-associated dental problems. *If a patient is having recurrent problems due to their RA or medication, please discuss this with their rheumatologist or GP.*

### Concurrent medications

As for any patient, it is important to take and keep updated a thorough medical/surgical history with a full medication list. Be aware of drug interactions when prescribing (particularly antibiotics); if in doubt consult the British National Formulary.<sup>101</sup> If prescribing NSAIDs, check what the patient is already taking and assess toxicity risk.<sup>94</sup> Some RA patients take oral bisphosphonates for the prevention or treatment of osteoporosis; therefore, there is a small risk (estimated at 0.5%) of osteonecrosis of the jaw following dentoalveolar surgery.<sup>102</sup> This risk can be increased with concomitant use of corticosteroids, and in some cases bisphosphonates may need to be stopped at least two months prior to surgery.<sup>102</sup> Biologic DMARDs should be stopped much before major surgical procedures (according to the half-life of drug) so as not to increase infection risk.<sup>6</sup> Conventional DMARDs may need to be stopped prior to procedures which last over an hour and it is recommended corticosteroid exposure be minimised prior to surgery.<sup>103</sup> *Therefore it is important to consult with the patient's rheumatologist well in advance of any planned invasive procedures and to follow up the patient postoperatively.*

### Antibiotic and steroid cover

The National Institute for Health and Care Excellence's recently updated guidance is that patients considered at high risk of infective endocarditis do not routinely require antibiotic prophylaxis for dental procedures, though this may be appropriate in individual cases.<sup>104</sup> *If in doubt, consult with the patient's cardiologist.*

Latest guidelines from the British Society

of Rheumatology state that pre-operative increase in steroid dose, for adrenal crisis prevention, is no longer routinely required.<sup>103</sup> However, if a patient shows signs of adrenal crisis (vomiting, abdominal pain, syncope, low blood pressure, hypoglycaemia, confusion) during a procedure, *seek immediate emergency medical attention.*<sup>94</sup>

### Co-morbidity detection

Dental professionals should be aware of the potential co-morbidities with RA and *refer patients on to an appropriate medical professional for further investigation (copying in the patient's rheumatologist and GP) if they detect anything of concern.*

**IT IS IMPORTANT TO NOTE THAT**

**XEROSTOMIA MAY BE REPORTED**

**BY PATIENTS BEFORE OBVIOUS SIGNS OF**

**HYPOSALIVATION ARE VISIBLE IN THE MOUTH.'**

### Conclusion

RA is a multi-faceted disease which can be complex to manage as co-morbid conditions are frequently present. Dental complications can arise associated with RA or its treatment. It is important that the dental team are aware of them so these patients can be successfully managed in general dental practice, with early intervention to prevent a further decline in their quality of life. Simple adjustments can be made to make dental visits more comfortable for patients with this long-term condition.

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**‘DUE TO PAIN, IMPAIRED HAND FUNCTION**

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**'DENTAL PROFESSIONALS SHOULD BE**

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**WITH RA AND REFER PATIENTS ON IF THEY**

**DETECT ANYTHING OF CONCERN.'**

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