

RHEUMATOID ARTHRITIS: A REVIEW AND SUGGESTED DENTAL CARE CONSIDERATIONS

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ABSTRACT

Background. Rheumatoid arthritis, or RA, is a chronic multisystem disease of presumed autoimmune etiology. It is estimated that arthritis and other rheumatic conditions affect 42.7 million Americans. Medical complications due to RA and its treatment may affect the provision of oral health care.

Methods. The authors undertook an extensive review of the English literature relating to RA and dental care. They used primarily MEDLINE searches, which included such key words as “rheumatoid arthritis” and “dental care” and subsequent appropriate subheadings. While the MEDLINE search spanned the years from 1975 to the present, the most recent literature was prioritized. Appropriate medical and dental textbooks were also used. The authors extrapolated information from selected texts based on its relevance to dentistry, oral health and the role of the dental provider in the overall treatment of RA patients.

Results. The authors reviewed nearly 200 articles and seven textbooks. Their determination of the texts’ relevance to oral health care was based on content, significance, quality, journal in which articles were published and year of publication. Major features of RA—including its diagnosis, pathophysiology, clinical features and medical treatment—were

identified, as well as complications due to treatment modalities and various related oral manifestations and conditions.

Conclusions. Medical complications due to RA and its treatment can affect oral health care. Oral health care providers need to recognize and identify modifications of dental care based on the medical status of patients with RA. Furthermore, oral health care providers play an important role in the overall care of these patients as it relates to early recognition, as well as control of the disease.

Clinical Implications. In most patients with RA, the condition will necessitate few or no changes in routine dental care. However, considerations include the patient’s ability to maintain adequate oral hygiene, xerostomia and its related complications, the patient’s susceptibility to infections, impaired hemostasis, and untoward drug actions and interactions. Patients with RA may require antibiotic prophylaxis owing to joint replacement and/or immune suppression, glucocorticosteroid replacement therapy and modifications in oral hygiene procedures. Intra- and extraoral conditions such as ulcerations, gingival overgrowth, disease-associated periodontitis and temporomandibular pathology also need to be recognized.

Rheumatoid arthritis, or RA, was first described clinically in a 1800 doctoral thesis by Landre-Beauvais, a French medical student, who called the condition “primary aesthenic gout.” Sir Alfred Garrod established the distinction between RA and gout in 1859 and gave the condition its present name.¹ Still today, however, many rheumatologists have suggested that RA is more

likely a group of diseases in which chronic polysynovitis is a major manifestation.²

Arthritis and other rheumatic conditions involving chronic joint symptoms affect 42.7 million Americans, costing \$65 billion per year.^{3,4} Nearly three million Americans have the specific diagnosis of RA. Interestingly, approximately 40 percent of people with chronic joint symptoms have not

contacted a physician for a diagnosis.⁵ Women are about three times more likely to be affected than men, and 80 percent of people with RA develop signs and symptoms of the disease between the ages of 35 and 50 years.^{6,7} The classic characteristics of this disease are bilateral and symmetric chronic inflammation of the synovium, a condition known as synovitis. This inflammatory response particularly affects small joints of the upper and lower extremities, and it often leads to the deterioration and eventual destruction of articular cartilage and juxta-articular bone, as well as to an inflammatory process surrounding tendons, all of which frequently result in deformities of the affected joints.

In addition to the typical pattern of inflammation, patients with RA may experience systemic manifestations such as fatigue, loss of appetite, weakness and vague musculoskeletal pain. Recent studies have established a connection between RA and an increased risk of premature mortality due to infections, hematopoietic malignancies, cardiovascular diseases, renal diseases and/or complications from treatment.⁸ While patients with RA appear to die as a result of the same causes affecting the general population, the increased mortality in people with RA is proportional to the severity of the course of their disease.⁹

Successful management of this condition requires a multifaceted and multidisciplinary approach to treatment. Treatment modalities include systemically administered drugs, local injections of corticosteroids, physical therapy, occupational therapy, psychological

counseling, patient education and surgical intervention.

PATHOPHYSIOLOGY

The cause of RA is not known, although its etiology appears to be multifactorial and may involve infectious, genetic, endocrine and immune participation.^{6,7} Rheumatoid arthritis is believed to be a T lymphocyte-driven disease in which a sudden influx of T cells into the affected joint(s) is followed by an increased number of macrophages and fibroblasts, drawn by the release of cytokines, par-

Fifty percent of people with rheumatoid arthritis become unable to work within the decade after the onset of disease.

ticularly interleukin-1, or IL-1, and tumor necrosis factor- α , or TNF- α . This cytokine release and subsequent migration of cells is thought to be responsible for the chronic inflammation, and characteristic destructive changes in rheumatoid joints.^{6,10} The cause of the initial T-cell influx is not known, but several infectious agents—including both bacteria such as streptococci and mycoplasma as well as viruses such as parvovirus, Epstein-Barr virus and retroviruses—have been suggested.

Synovial joints are composed of articular cartilage, synovial fluid and a synovial membrane. The synovial membrane is the area of the joint infiltrated by the T cells in a rheumatoid attack and is the site of the subsequent immune response. The severity and progression of RA-

provoked synovitis depends on the local accumulation and activation of cytokine-releasing cells, which appear to regulate the growth, differentiation and activity of other cells involved in inflammatory and immunological reactions in the rheumatoid joint. The hypertrophied, inflamed synovial tissue that covers and extends into the cartilaginous areas of the joint with fingerlike processes is defined as the pannus. Cytokines increase the permeability of blood vessel walls, facilitating the migration of white blood cells into joint spaces that then become sites of inflammation. Cytokine release also leads to the proliferation of fibroblasts, synovial cells, increased prostaglandin and matrix-degrading protease activity and, ultimately, the resorption of bone. IL-1, a cytokine secreted primarily by macrophages, is one of the key mediators of local inflammation, tissue damage, immunologic reactions and bone resorption.¹¹ Additionally, it is implicated in many of the systemic manifestations of RA, particularly malaise, fever, anemia and elevated serum acute-phase reactants.¹² TNF- α , another key cytokine that often acts synergistically with IL-1, also promotes the cartilage and bone erosion that leads to RA-characteristic pathological joint alteration.¹³

Joint pathology is pervasive in patients with RA. The greatest rate of joint damage occurs in the first two years of the disease, when more than 50 percent of patients have radiographic evidence of joint damage. Within three years, nearly 70 percent of affected people will exhibit radiographic damage.^{14,15} Somewhat less than

10 percent of patients report single, nonrecurrent episodes of RA, or mild but recurring cases, while two-thirds of patients exhibit a clinical pattern of waxing and waning disease over many years.^{16,17} The general progression of RA leads to an increasing disability and loss of functional capacity; 50 percent of people with RA become unable to work within the decade after the onset of disease.¹⁸

There are extra-articular manifestations of RA as well, including rheumatoid nodules, rheumatoid vasculitis, interstitial lung disease, pericardial disease, episcleritis and scleritis, Felty's syndrome and Sjögren's syndrome. Felty's syndrome, a rare condition affecting 1 to 2 percent of people with RA, is characterized by chronic RA, splenomegaly, neutropenia and sometimes anemia or thrombocytopenia.¹⁹ These patients can have neutrophil counts of less than 1,000 cells per microliter as well as defective neutrophil function.²⁰ Sjögren's syndrome is a chronic autoimmune disease characterized by lymphocytic infiltration of the exocrine glands resulting in xerostomia, dry eyes and joint pain. When observed in association with RA, it is called "secondary Sjögren's syndrome."^{7,21}

DIAGNOSIS

In 1987, the American Rheumatism Association (now the American College of Rheumatology, or ACR) revised the criteria it had set in 1958 to create a better model for the diagnosis of RA (Table 1).^{22,23} There are no specific laboratory tests to diagnose RA. Rheumatoid factors—immunoglobulin M, or IgM, antibodies directed against other immunoglobulins—are

TABLE 1

1987 REVISED CRITERIA FOR THE CLASSIFICATION OF RHEUMATOID ARTHRITIS.*

GUIDELINES	
<ul style="list-style-type: none"> Four of seven criteria must be satisfied for a diagnosis. Criteria 1 through 4 must be present for at least six weeks. Criteria 2 through 5 must be observed by a physician. Designation as classic, definite or probable rheumatoid arthritis, or RA, is not to be made. 	
Criteria	Definition
1. Morning Stiffness	Stiffness in and around the joints, lasting at least one hour before maximal improvement
2. Arthritis of Three or More Joint Areas	Soft-tissue swelling or fluid (but not just bony overgrowth) in at least three joint areas simultaneously (the 14 possible joint areas are right or left proximal interphalangeal, or PIP; metacarpophalangeal, or MCP; wrist; elbow; knee; ankle; and metatarsophalangeal, or MTP, joints)
3. Arthritis of Hand Joints	At least one area swollen (as defined in criterion 2) in a wrist, MCP or PIP joint
4. Symmetric Arthritis	Simultaneous involvement of the same joint areas (as defined in criterion 2) on both sides of the body
5. Rheumatoid Nodules	Subcutaneous nodules over bony prominences, extensor surfaces or juxta-articular regions
6. Amount of Serum Rheumatoid Factor	Demonstration of abnormal amounts of serum rheumatoid factor by any method for which the result has been positive in less than 5 percent of normal control subjects
7. Radiographic Changes	Changes typical of RA on posteroanterior hand and wrist radiographs, which must include erosions or unequivocal bony decalcification localized in or most marked adjacent to the involved joints
* Adapted from Arnett and colleagues. ²³	

TABLE 2

TOXICITIES OF ANTIRHEUMATIC DRUGS.*	
DRUG	TOXICITIES
Methotrexate	Gastrointestinal symptoms, stomatitis, rash, alopecia, infrequent myelosuppression, hepatotoxicity, rare but potentially life-threatening pulmonary toxicity
Injectable Gold Salts	Rash, stomatitis, myelosuppression, thrombocytopenia, proteinuria
Oral Gold Salts	Same as those of injectable form but less frequent; frequent diarrhea
D-penicillamine	Rash, stomatitis, dysgeusia, proteinuria, myelosuppression, infrequent but serious autoimmune disease
Cyclosporine	Renal impairment, hypertension, gingival overgrowth
Nonsteroidal Anti-inflammatory Drugs	Gastrointestinal symptoms, including indigestion, ulceration, hemorrhage, small-bowel ulceration, stomatitis; renal abnormalities; neurological abnormalities; pulmonary abnormalities; dermatologic abnormalities; hepatic abnormalities; displacement of protein-bound drugs; possible systemic complications
* Adapted from American College of Rheumatology Ad Hoc Committee on Clinical Guidelines ²⁷ and Lehmann and colleagues. ⁵⁰	

found in more than two-thirds of adult patients with RA, but they are not specific to RA and are found in patients with a number of other conditions. Additionally, 5 percent of otherwise healthy people have circulating rheumatoid factors.⁷ While the presence of rheumatoid factor is not useful as a screening test, high levels in combination with a suggestive clinical presentation of RA are prognostic for more severe disease with extra-articular manifestations.

The latest set of criteria was developed through the study of

patients who already had been diagnosed with RA and had a median disease duration of 7.7 years.²² Therefore, they are more helpful in evaluating those with well-established symptoms, and often are difficult to apply to people in early stages of the disease.²² Evaluations of specific patterns of pain in suspected cases have been able to predict RA with a 70 percent probability at early stages of the disease.²⁴

TREATMENT

The objective of RA therapies is

to restore or at least maintain quality of life by relieving pain, reducing joint inflammation and preventing joint destruction and deformity.²⁵ The challenge for physicians in treating RA is preventing early stages of the disease from progressing to stages of severe erosion and joint deformity.²⁶ Currently available antirheumatic drugs control established RA only partially. They rarely induce long-term remission and seldom affect the progression of joint destruction.²⁷ Additionally, many of these drugs have serious side effects that contraindicate their use for early-stage RA and interfere with long-term therapeutic use for more severe RA cases^{28,29} (Table 2). Physical therapy, exercise, use of orthotic devices and, in severe cases, surgery all are factors in treatment planning.⁷

To prevent erosive damage by progressive RA, the condition must be diagnosed early and therapy begun promptly, ideally within two months of disease onset.⁶ Nonsteroidal anti-inflammatory drugs, or NSAIDs, are the current mainstream "first-line" treatment, although this protocol is now criticized by many physicians who are arguing for more aggressive early treatment of RA. However, while NSAIDs often are effective in controlling symptoms of RA, they do not alter the course of the disease. Corticosteroids, another option, have both anti-inflammatory and immunosuppressive effects. This type of drug can provide effective relief by decreasing circulating monocytes, reducing macrophage phagocytosis and IL-1 secretion, inhibiting collagenase and lysosomal enzyme release, and slowing

TABLE 3

RHEUMATOID ARTHRITIS AGENTS.*		
AGENT	DOSING	COST PER MONTH IN DOLLARS†
Auranofin	3 milligrams per mouth, or PO, twice daily	79
Aurothioglucose	50 mg intramuscularly once every two weeks	28
Azathioprine	150 mg PO once daily	105
Celecoxib	100 mg PO twice daily	86
Cyclophosphamide	150 mg PO daily	300
Cyclosporine	2 mg/kilogram PO twice daily	475
D-penicillamine	750 mg PO once daily	86
Etanercept	25 mg subcutaneously twice weekly	1,100
Gold Sodium Thiomalate	50 mg once every two weeks	18
Hydrochloroquine	200 mg PO twice daily	64
Leflunomide	20 mg PO once daily	245
Methotrexate	15 mg PO once daily	75
Sulfasalazine	1,000 mg PO three times daily	52
* Adapted from Dunn and Small. ⁵⁷		
† Based on average wholesale price in December 1998.		

prostaglandin and leukotriene synthesis. However, corticosteroid therapy is limited in its usefulness because of its severe side effects, which may include osteoporosis, muscle weakness, glucose intolerance, cataracts and sex hormone imbalances.⁷ In cases of long-term use of glucocorticosteroids, the drugs usually are given only every other day to reduce potential side effects.

“Second-line” or disease-modifying antirheumatic drugs, or DMARDs, whose mechanisms of action as a group are predominantly unknown, include gold, sulfasalazine, hydroxychloroquine, D-penicillamine, azathioprine and a recently approved drug, leflunomide (Arava, Hoechst Marion Roussel Inc.) (Table 3). While NSAIDs and

glucocorticoids treat primarily symptoms, DMARDs have the potential to reduce disease ac-

The primary goal of the latest treatment protocols has been aggressive early control of inflammation, based largely on the finding that significant joint damage does occur in the early stages of rheumatoid arthritis.

tivity and/or prevent joint damage.²⁷ These second-line agents can have considerable toxicity. Therefore, their use requires

frequent patient monitoring and ultimately limits their therapeutic value, as many patients are able to tolerate the drugs for only one or two years before they are forced to discontinue the treatment.^{17,30}

Methotrexate has become a popular treatment choice recently because of its immunosuppressive and anti-inflammatory effects.³¹ This drug has been proven effective. Also, it is tolerable as a long-term treatment, particularly as folic acid supplements can be used to ameliorate many of its toxic side effects, which may still include gastrointestinal distress, stomatitis, thrombocytopenia, bone marrow suppression, pneumonitis and/or pulmonary lesions, hepatic fibrosis/cirrhosis and renal toxicity. Well more

INDICATIONS FOR ANTIBIOTIC PROPHYLAXIS FOR DENTAL PATIENTS WITH RHEUMATOID ARTHRITIS AND TOTAL JOINT REPLACEMENTS.*

DENTAL PROCEDURES FOR WHICH ANTIBIOTIC PROPHYLAXIS IS INDICATED

- Dental extractions
- Periodontal procedures
- Dental implant placement and reimplantation of avulsed teeth
- Endodontic instrumentation or surgery only beyond the apex
- Initial placement of orthodontic bands (not brackets)
- Intraligamentary local anesthetic injections
- Prophylactic cleaning of teeth or implants when bleeding is anticipated

SUGGESTED ANTIBIOTIC PROPHYLAXIS REGIMENS

Note: No second doses are recommended for any of these regimens.

- Patients not allergic to penicillin: cephalexin, cephadrine or amoxicillin, 2 grams orally, one hour before dental procedure
- Patients not allergic to penicillin and unable to take oral medications: cefazolin (1 g) or ampicillin (2 g) intramuscularly or intravenously, one hour before dental procedure
- Patients allergic to penicillin: clindamycin, 600 milligrams orally, one hour before dental procedure
- Patients allergic to penicillin and unable to take oral medications: clindamycin, 600 mg intravenously, one hour before dental procedure

* Adapted from American Dental Association and American Academy of Orthopaedic Surgeons.⁵³

than one-half—64 percent—of people who use methotrexate are able to continue its use for five years or more.^{32,33}

Signs and symptoms of RA typically recur when second-line drug therapies are discontinued. Because most second-line therapy (with the exception of methotrexate therapy) must be discontinued after less than two years owing to toxicity or loss of efficacy, recurrence and long-term control of RA is a problem.³⁴ The primary goal of the latest treatment protocols, however, has been aggressive

early control of inflammation, based largely on the finding that significant joint damage does occur in the early stages of RA, particularly in the first two years of the disease.^{15,27} New treatment strategies have sought to maximize long-term efficacy of second-line drugs by introducing them earlier and giving them in combinations.³⁵ In controlled trials, however, multiple-agent treatments have failed to demonstrate greater efficacy than single agents, and the toxicity of some combinations has proven to be signifi-

cantly greater than that of single agents. Combinations of methotrexate and cyclosporine have shown some promise.² Moreover, there is little evidence supporting the effectiveness of aggressive drug therapy in altering the long-term consequences of RA.³⁵

One of the latest and more novel approaches to treatment is cytokine therapy, which could involve the inhibition of cytokine synthesis, inhibition of cytokine release, inhibition of cytokine action and/or inhibition of cytokine intracellular signaling pathways. While some of these modalities are showing promising clinical success, it remains uncertain how effective blocking single cytokines can be in inhibiting the complex cytokine-induced pathology of RA.³⁶

Surgical intervention is used in cases of unacceptable pain and limitation or loss of function due to severe joint damage. The most successful procedures include arthroplasties and total joint replacements involving the hips, knees and shoulders.^{7,27}

ORAL MANIFESTATIONS AND CONDITIONS

As mentioned above, long-term use of methotrexate and other antirheumatic agents such as gold, D-penicillamine and NSAIDs can cause stomatitis. Cyclosporine may cause gingival overgrowth. Additionally, most patients with RA will exhibit some temporomandibular joint, or TMJ, involvement during the course of the disease. Involvement of the TMJ results from granulomatous involvement of the articular surface of the synovial membrane, which leads to destruction of the underlying bone. Symptoms are characteristic of TMJ dysfunction.³⁷

Radiographic findings include narrowed joint spaces, flattened condyles, erosions, subchondral sclerosis, cysts and osteoporosis.^{38,39} Severe arthritic deterioration of the TMJ may be accompanied by a high incidence of upper-airway obstruction.⁴⁰

Patients with longstanding active RA may have an increased incidence of periodontal disease, including loss of alveolar bone and teeth.^{41,42} This has been a neglected feature of RA. Similarities in host immune response between RA and periodontal disease have been reported, involving reduced cellular and enhanced humoral activity.^{43,44} While a protective influence of NSAIDs on gingivitis and periodontitis has been reported because of their reduction of inflammation and, therefore, of subsequent loss of bone, there is little knowledge regarding the impact of second-line agents on periodontal disease.⁴⁵ It does not appear that inadequate oral hygiene resulting from functional impairment is a primary factor in periodontal disease.⁴¹

Patients with secondary Sjögren's syndrome have chronic xerostomia. A recent study of 604 people with RA indicated a decrease in salivary flow in 43 percent of patients.⁴⁶ Furthermore, the risk of developing reduced salivary flow increased with severity of the disease. This leads to multiple oral problems, including difficulty in swallowing food, difficulty in speaking, oral soreness and burning (which may be due to oral candidiasis), intolerance to spicy foods, problems in wearing dentures and an increase in caries.^{7,21,47,48} Caries in these patients may progress despite excellent regular home oral care,

TABLE 4

GUIDELINES FOR CARIES PREVENTION FOR PATIENTS WITH SJÖGREN'S SYNDROME AND/OR REDUCED SALIVARY FLOW.*

THERAPY	GUIDELINES
Treatment Planning	Four-month recall visits; frequent, high-quality bitewing radiographs; conservative restorations based on patient hygiene, compliance with fluoride use and caries susceptibility
Personal Oral Hygiene Instruction	Brush at least twice daily with a fluoride dentifrice; floss or, space permitting, use an interproximal brush
Treatment of Xerostomia	Water, artificial saliva, sugarless gum or mints, 5 milligrams of pilocarpine hydrochloride 3 to 4 times a day (note that adverse reactions to pilocarpine hydrochloride include increased sweating in 40 percent of patients)
Dietary Instruction	Limit between-meal ingestion of fermentable carbohydrates and encourage use of noncariogenic sweetening agents such as acesulfame K, aspartame, saccharin, sorbitol or xylitol
Office Fluoride Treatment	1.23 percent fluoride acidulated phosphate fluoride, or APF, gel for four minutes in a tray; or a 2.25 percent fluoride varnish applied directly to the teeth, four times a year
Office Chlorhexidine Treatment	Application of a 1 percent chlorhexidine gel for five minutes or a high-concentration chlorhexidine varnish applied directly to the teeth (neither yet available in the United States)
Home Fluoride Treatment	Daily five-minute application of 1.1 percent sodium fluoride, or NaF, or of APF gel in a custom tray; or daily 0.05 percent NaF rinse for one minute is an alternative for patients unable to tolerate the gel owing to gagging or nausea; use of extremely-high-fluoride-content toothpaste once daily
Home Chlorhexidine Treatment	If on three-month recall, the <i>Streptococcus mutans</i> count exceeds 1×10 colony-forming units per milliliter of saliva, institute a 30-second rinse twice daily with 0.12 percent chlorhexidine gluconate for two weeks

* Adapted from Newbrun,⁴⁷ Atkinson and Fox,⁴⁸ Atkinson and Wu,⁴⁹ Fox and colleagues.⁵⁵

SUMMARY: DENTAL MANAGEMENT OF THE RHEUMATOID ARTHRITIS PATIENT.

BEFORE TREATMENT

- Administer antibiotic prophylaxis because of joint replacement when indicated
- Administer antibiotic prophylaxis because of immune suppression when indicated
- Administer glucocorticosteroid replacement therapy when indicated
- Assess potential impairment of hemostasis

DURING TREATMENT

- Recognize and address ulcerations and gingival overgrowth due to medications
- Recognize and address xerostomia-associated complications
- Recognize and address disease-associated periodontitis

AFTER TREATMENT

- Avoid untoward drug actions and interactions when choosing postoperative medications
- Improve and modify oral hygiene instruction

use of fluoride and avoidance of cariogenic foods. Unexplained dental caries, especially in root and incisal sites, may be the first apparent clinical sign in Sjögren's syndrome.⁴⁹

DENTAL MANAGEMENT

It is essential that the dentist keep himself or herself updated as to the drugs the patient with RA is currently receiving, their possible side effects and interactions with other drugs. The most common adverse effects involved with NSAIDs include the gastrointestinal and renal systems, and both are dose-related. Before prescribing additional NSAIDs, the clinician must assess the RA patient's current medication schedule to avoid toxic levels, especially in patients with a history of renal

impairment or peptic ulcers.⁵⁰ GI-protective agents such as the prostaglandin analog misoprostol may help alleviate these side

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effects.²⁷ NSAIDs are known to displace protein-bound drugs as well as impair renal function, both of which can increase free levels of methotrexate, which is 50 to 60 percent albumin-bound

and eliminated in large part in the urine by active secretion in the proximal tubules of the kidneys.⁵¹ Although it is somewhat inconclusive, there is evidence for a greater possibility of infections in patients using methotrexate owing to immunosuppression. While stomatitis is considered a relatively minor adverse effect of methotrexate use, folic acid may help reduce its severity.⁵¹

With long-term glucocorticosteroid use, secondary adrenal insufficiency is a potential problem. Replacement therapy for adrenally suppressed people may be necessary to prevent cardiovascular collapse, as their response to surgical stress may include a precipitous drop in blood pressure. In such cases, an intramuscular or intravenous injection of hydrocortisone may be necessary. Long-lasting local anesthetics (such as bupivacaine) and postoperative pain medications should be used with these patients, as well as mild sedatives in more apprehensive patients.⁵²

According to the guidelines recently published by the American Dental Association and the American Association of Orthopaedic Surgeons,⁵³ it is recommended that patients with severe RA who have had joints surgically replaced with prosthetic joints may require prophylactic antibiotic therapy before invasive dental procedures (Box, "Indications for Antibiotic Prophylaxis for Dental Patients With Rheumatoid Arthritis and Total Joint Replacements"). Patients with RA who have upper-airway obstruction resulting from TMJ dysfunction may pose difficulty in intubation.³⁷ In addition, depending on the severity

of the TMJ dysfunction, the patient may require a soft diet restriction. These patients may derive long-term benefits from local physical therapy of the stomatognathic system.⁵⁴

Patients with Felty's syndrome are at an increased risk of developing infection owing to their neutropenia and impaired hemostasis owing to their thrombocytopenia.²⁰ Patients with Sjögren's syndrome may require additional personal oral care instruction, dietary instruction and modifications, home/clinical fluoride therapy, home/clinical chlorhexidine therapy, treatment for their xerostomia, more frequent recall visits and radiographs and more conservative treatment plans⁴⁸ (Table 4). Pilocarpine hydrochloride (Salagen, MGI Pharma Inc.) was recently approved in the United States for treatment of Sjögren's syndrome in patients who are experiencing hyposalivation and have remaining functional salivary tissue.⁵⁵

Additional concerns when treating dental patients with RA are patient comfort and patient education. According to the severity of the patient's RA, certain considerations should be addressed. The dentist should be conscious of the patient's physical comfort level in the dental chair: altering the chair's position, allowing the patient to shift positions, using pillows and other such aids, and scheduling shorter appointments all should be considered. Home oral hygiene procedures may present a challenge to the patient with RA because of reduced manual dexterity. To enhance traditional dental hygiene practices, floss holders, electric toothbrushes, irrigating

devices, and chlorhexidine and fluoride rinses all can be helpful to RA patients experiencing symptoms in the hands and wrists.^{56,57} Specially designed toothbrushes are also available for patients with RA.

CONCLUSION

For most patients with RA, few or no changes will be required in standard dental treatment (Box, "Summary: Dental Management of the Rheumatoid Arthritis Patient"). However, it is important to assess the status of the patient's condition carefully, as even mild cases of RA may adversely affect the patient's ability to maintain good oral hygiene, especially in patients with xerostomia. Associated syndromes may contribute to a patient's susceptibility to infections and impaired hemostasis. A proper review of the patient's medication history will allow for more accurate differential diagnoses of oral lesions and will minimize complications with drug interactions or overdoses. Practitioners specifically need to be aware of potential adrenal insufficiency in patients receiving long-term glucocorticosteroid therapy. A further concern is patients who have had joint replacement, for whom antibiotic prophylaxis is indicated.

As with many other chronic conditions, early intervention can reduce the severity of the disease. Thus, dental health-care workers play an important role in recognizing signs and symptoms of RA and in advising patients to seek medical care. ■

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