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Understanding the effects of rheumatoid arthritis

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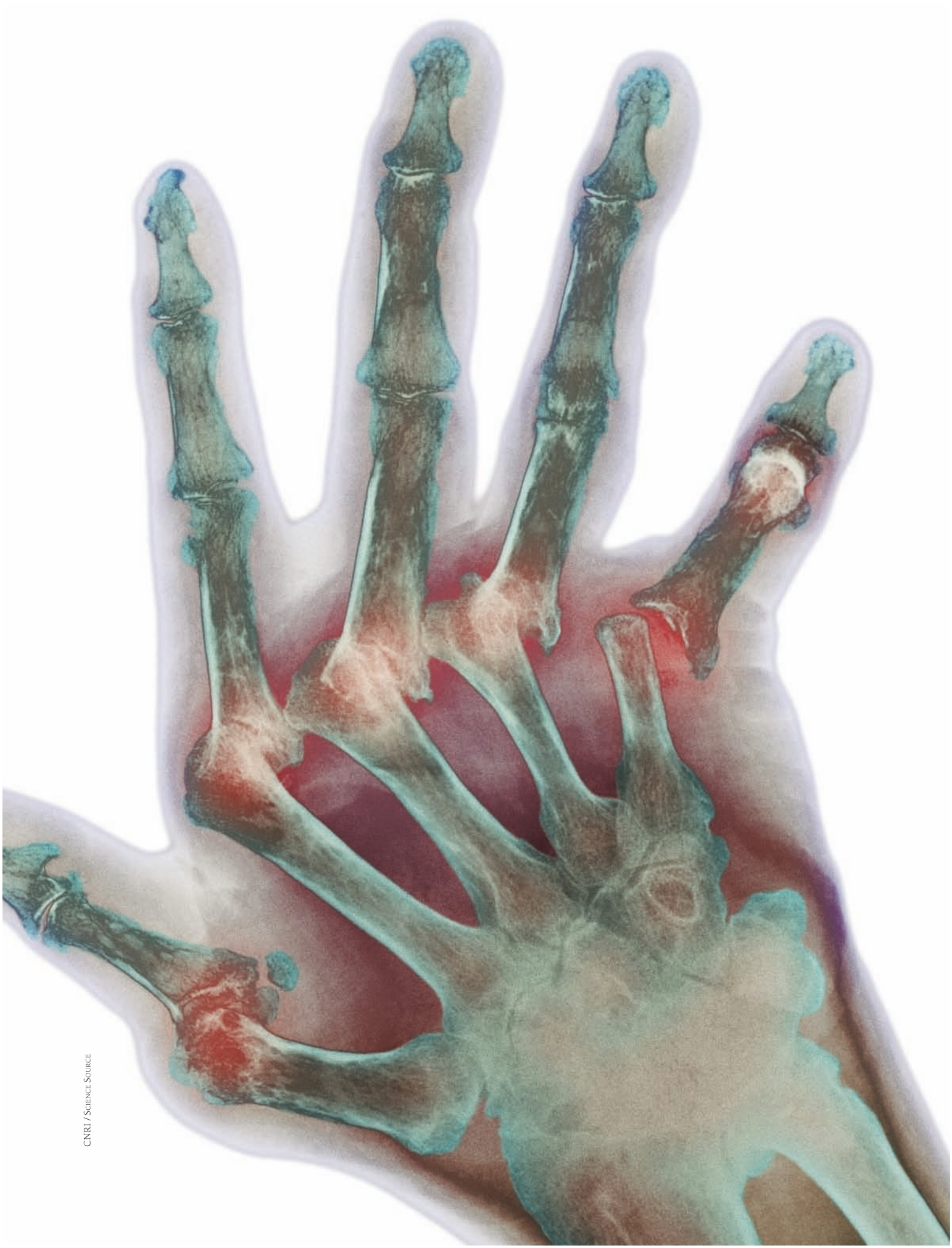
RHEUMATOID ARTHRITIS (RA) is a chronic, systemic autoimmune disease involving inflammation and degeneration of the joints that affects an estimated 1% of people worldwide.[1] RA is an incurable disease, and the debilitating signs and symptoms usually negatively affect a person's quality of life. This article will discuss RA, including how it's diagnosed, and review appropriate nursing care to manage the disease.

Understanding RA

RA is an abnormal immune response that leads to synovial inflammation and destruction of joints. It may be initiated by the activation of CD4+ helper T cells, the local release of inflammatory mediators and cytokines (such as tumor necrosis factor [TNF] and interleukin [IL]-1) that destroy joints, and the development of antibodies that are directed against joint-specific and systemic autoantigens.[2]

An antibody called the rheumatoid factor (RF), which reacts with immunoglobulin G to form immune complexes, is found in 70% to 80% of patients with RA. However, the role of the autoimmune process in joint destruction is still unknown. At the cellular level, neutrophils, macrophages, and lymphocytes are attracted to the area; the neutrophils and macrophages phagocytize the immune complexes and release lysosomal enzymes that can cause destructive changes in the joint cartilage (see *Pathophysiology of RA*). The inflammatory response then attracts additional inflammatory cells, continuing the process. Synovial cells and subsynovial tissues undergo reactive hyperplasia, and joint edema results from



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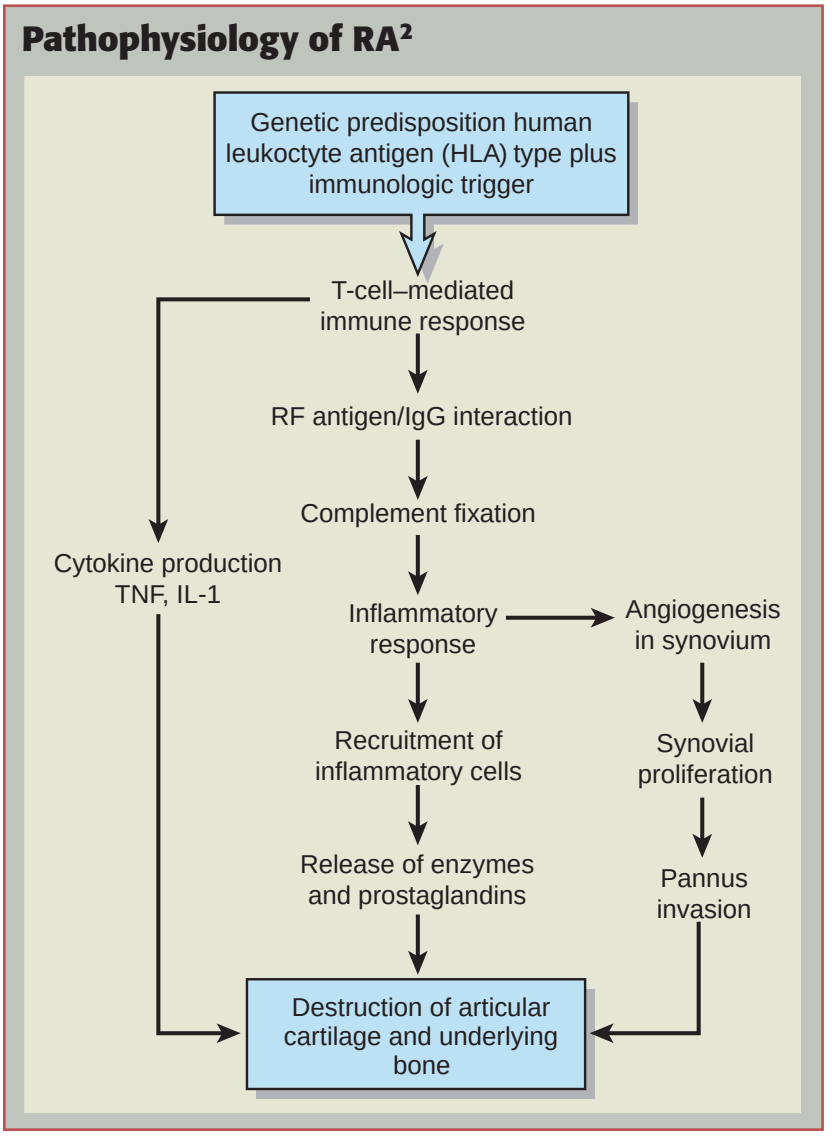
an increased blood flow and capillary permeability that's part of the inflammatory process.[2]

The development of an extensive network of new blood vessels in the synovial membrane contributes to the advancement of rheumatoid synovitis in RA. The pannus (destructive vascular granulation tissue), which differentiates RA from other forms of inflammatory arthritis, extends from the synovium to an area of unprotected bone at the junction between cartilage and subchondral bone called the "bare area" (see *A closer look at RA*). The inflammatory cells found in the pannus destroy adjacent cartilage and bone, and the pannus will eventually develop between the joint margins. This leads to reduced joint motion and the possibility of ankylosis (fusion of joints). As RA progresses, joint inflammation and structural changes lead to joint instability, muscle atrophy from disuse, stretching of the ligaments, and involvement of the tendons and muscles. These destructive changes are permanent.[2]

Though the exact cause is unknown, a familial history of RA coupled with an activation of T-cell mediation in response to some sort of immunologic trigger (such as a microbial agent) is believed to play a part in development of the disease. Though RA may affect people in all age-groups, the incidence increases with age, with women being more commonly afflicted than men.[2-4] (See *Risk factors for RA*.)



Recognizing RA

Clinical manifestations of RA vary depending on the severity of the disease. Characteristic signs and symptoms include edema and pain in the affected joints along with warmth to touch, erythema, stiffness (especially morning stiffness), and loss of joint function. Palpated joints have a boggy or spongy feel, and fluid can often be aspirated from an affected joint. Initial affected areas typically include the small joints of the hands, wrists, and feet. As the disease progresses, other joints, including the knees, shoulders, hips, ankles, spinal processes, and temporomandibular joints, become affected.[2-5] Deformities of the hand and feet are common, including partial dislocation (subluxation) of the joints where one bone moves over another bone. (See *Hand deformities in RA*.) Specific hand deformities include:

- Boutonnière deformity (flexion of the proximal interphalangeal [PIP] joint and hyperextension of the distal interphalangeal [DIP] joint)
- swan-neck deformity (hyperextension of the PIP and flexion of the DIP)
- ulnar deviation or "ulnar drift" with subluxation of the metacarpophalangeal joints.[2-5]

These deformities can reduce grip strength and decrease manual dexterity. In the foot, deformities may include hammertoe and claw toe. In the ankle, the disease often limits flexion and extension, which creates problems with ambulation.[2]

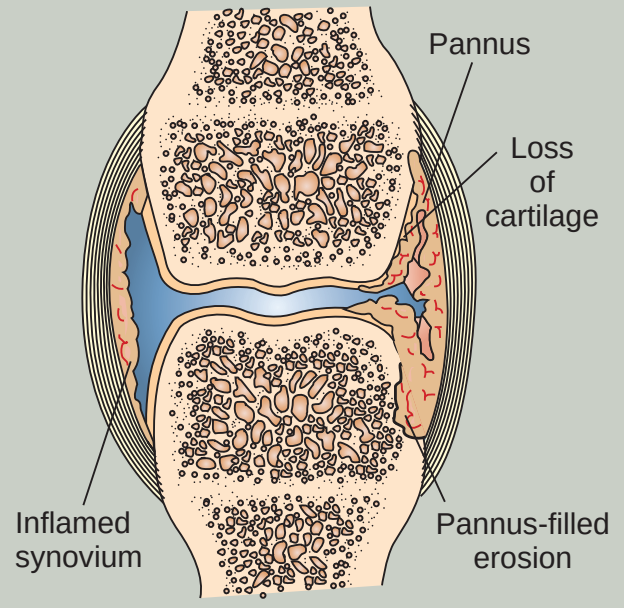
Onset of signs and symptoms is typically gradual, and symmetrical involvement of joints is a characteristic feature of RA.[6] Individuals with RA may try to protect these painful, swollen, and stiff joints by immobilizing them, which over time can lead to contractures and soft-tissue deformities involving the joints.

Although RA is primarily associated with involvement of the musculoskeletal system, the autoimmune "tissue-attacking" properties may affect other systems as well, including the skin, eyes, lungs, heart, blood vessels, salivary glands, central and peripheral nervous systems, and bone marrow.[7] Signs and symptoms of extra-articular disease in patients with RA include fever, weight loss, fatigue, muscle atrophy, lymphadenopathy, and signs and symptoms of Raynaud phenomenon. Patients may develop anemia, thrombocytosis, pleural effusions, pericarditis, endocarditis, and cardiac conduction abnormalities as well as neuropathies, scleritis, episcleritis, splenomegaly, and dry eyes and mucus membranes. In addition, about 25% of patients develop rheumatoid nodules, palpable nodules over bony prominences such as the elbow.[2-5]

Diagnosing RA

A diagnosis of RA requires a combination of subjective and objective findings along with lab test results. A thorough medical history is essential. During the physical exam, assess for bilateral/symmetric joint abnormalities, pain, edema, and limited joint movement; the presence of rheumatoid nodules; and signs of extra-articular disease. Other diagnoses such as polymyalgia rheumatica, osteoarthritis, infectious arthritis, and psoriatic arthritis should be ruled out. Lab testing should include assays for RF, which is present in 75% of RA patients.[4]

A closer look at RA[2]



Anticitrullinated protein antibodies are found in 60% to 70% of patients with RA.[8,9] The erythrocyte sedimentation rate (ESR) is elevated, while the red blood cell count and complement component 4 (C4) levels are low. C-reactive protein (CRP) levels may be normal or higher-than-normal and antinuclear antibody (ANA) titers may be positive.[10] Synovial fluid obtained by arthrocentesis appears cloudy, dark yellow, or milky with the presence of inflammatory biomarkers, including leukocytes and complement. Radiologic findings demonstrate distinctive bony erosions and narrowed joint spaces as the disease progresses.[4,5]

Treatment strategies

Because RA can't be cured, treatment focuses on preventing and limiting joint damage, loss of function, and other manifestations of the disease, in addition to managing pain. Encourage patients with RA to balance exercise and rest, eat a healthy diet high in vitamins, protein, and iron (for tissue building/repair), and take advantage of community support groups. Nonpharmacologic pain management strategies, such as relaxation techniques and heat and cold applications, may relieve some symptoms.

With disease progression, a more formalized exercise plan should be incorporated, including range-of-motion and muscle-strengthening exercises, and pacing of activities with rest periods for joint protection.[4,11] Other important interventions in all stages of RA include cardiovascular risk reduction and immunizations to decrease the risk of complications from drug therapies.[11]

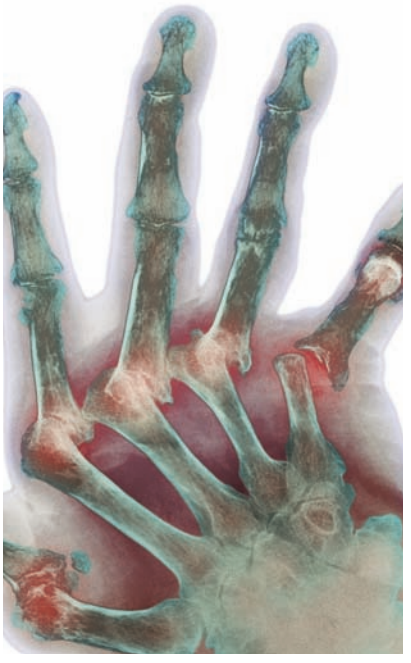
For many patients diagnosed with RA, a more aggressive treatment approach that includes the use of medication is the best course of treatment.[12] Evidence-based pharmacotherapy includes anti-inflammatory drugs, (nonsteroidal anti-inflammatory drugs [NSAIDs] and glucocorticoids),

and nonbiologic and biologic disease-modifying antirheumatic drugs (DMARDs).[13]

NSAIDs such as ibuprofen, naproxen, ketoprofen, and diclofenac are administered for rapid symptomatic relief of inflammation, pain, and stiffness associated with RA. Common adverse reactions to NSAIDs include gastric distress and possibly gastrointestinal (GI) bleeding, nausea, diarrhea, and constipation. In addition, NSAID use may be associated with various renal and cardiovascular adverse reactions such as edema, acute kidney injury, hypertension, myocardial infarction, and stroke. Monitor patients taking NSAIDs for symptom relief and adverse drug reactions.[12]

Glucocorticoids such as prednisone provide rapid control of inflammation. Possible adverse reactions to glucocorticoids include sodium retention, secondary adrenocortical insufficiency, GI perforation, osteoporosis, mood swings, and severe depression.[14]

All patients diagnosed with RA should be prescribed a DMARD such as hydroxychloroquine (HCQ) or sulfasalazine (SSZ) to suppress the body's overactive immune and inflammatory systems.[11] DMARDs should be started as soon as possible, ideally within 3 months of the onset of symptoms. DMARDs not only relieve RA symptoms but also slow progression of the disease.[9] The nonbiologic (or conventional synthetic) DMARDs most frequently used include HCQ, SSZ, and methotrexate (MTX).[11] Common adverse reactions to HCQ include blurred vision, abdominal pain, nausea, vomiting, anorexia, diarrhea, headache, skin rash, and pruritus. Bone marrow suppression is possible.[15]



Because RA can't be cured, treatment focuses on preventing and limiting joint damage and loss of function, and managing pain.

Common adverse reactions to SSZ include anorexia, headache, nausea, vomiting, and gastric distress.[16] Less frequent adverse reactions include skin rash, pruritus, urticaria, fever, and hemolytic anemia. Monitor the patient's complete blood cell count (CBC) results and assess for symptom relief.[12]

In patients with moderately to severely active RA, anti-inflammatory therapy with either an NSAID or glucocorticoid and DMARD therapy, generally with MTX is recommended. The most frequently reported adverse reactions to MTX include ulcerative stomatitis, leukopenia, nausea, abdominal distress, malaise, fatigue, chills and fever, dizziness, and decreased resistance to infection. More serious adverse reactions include hepatotoxicity and bone marrow suppression.[17] As the disease progresses, the immunosuppressant cyclosporine may be added to augment MTX.[4,12]

DMARDs take effect over weeks or months and aren't designed to provide immediate symptom relief. When taking a DMARD, patients should be carefully monitored for adverse drug reactions and taught about their prescribed medications, including adverse reactions and actions to take should they occur.

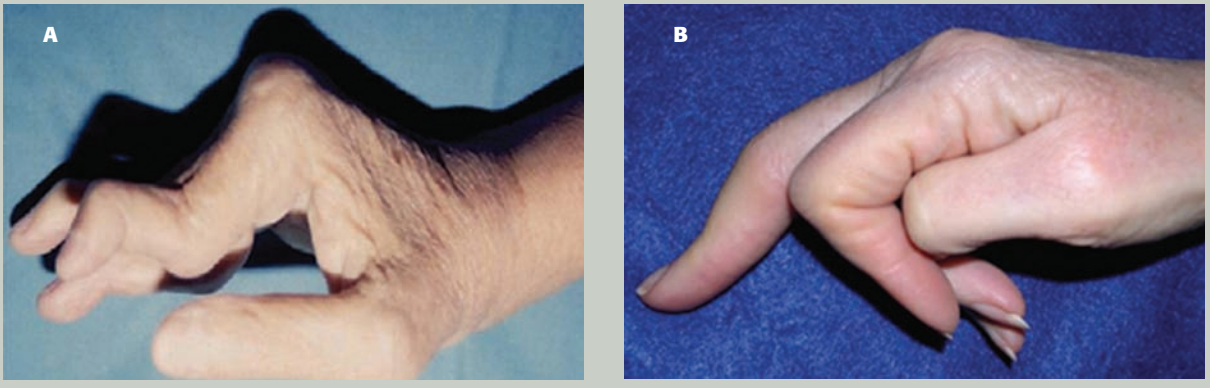
Some promising results have been shown with biologic DMARDs, which are produced by recombinant DNA technology. They target cytokines or their receptors or are directed against other molecules on the cell

Risk factors for RA[19]

The cause of RA is unknown, but is thought to be a result of environmental and genetic factors. Risk factors include:

- **Socio-demographics:** The incidence of RA is about two to three times higher in women than men. The onset of RA is highest in individuals over age 60.
- **Genetics:** Specific HLA class II genotypes are associated with increased risk of RA.
- **Smoking:** A history of smoking is associated with a 1.3 to 2.4 times increased risk of RA.
- **Reproductive and breastfeeding history:** Studies have found that women who have used oral contraceptives had a modest to moderate decreased risk of RA; women who have never had a live birth have a slight to moderately increased risk of RA; RA is less common among women who breastfeed; and women with irregular menses or early menopause have an increased risk of RA.

Hand deformities in RA



A. Rheumatoid arthritis of the hand demonstrating swan-neck deformity and volar subluxation of the MCP joints. B. Boutonnière deformity of the digit.
Source: Wiesel SW. *Operative Techniques in Orthopaedic Surgery*. 4th ed. Philadelphia, PA: Wolters Kluwer; 2015.

surface.[11] Biologic DMARDs include the non-TNF biologic agents, such as abatacept, and anti-TNF biologic agents such as adalimumab, etanercept, and infliximab.[13] When the body responds to infection and disease, specific cytokines (such as TNF-alpha and IL-1) promote an inflammatory response in the joints that contributes to joint destruction. Biologic DMARDs block this mechanism and inhibit the cytokine release, relieving inflammatory symptoms.[4,12] Common adverse reactions to biologic DMARDs include injection site reactions, abdominal pain, vomiting, and headache. Monitor the injection site for persistent erythema, pain, and edema, and assess the patient's response to therapy.[12]

Nursing interventions

Initial interventions in RA management include obtaining a comprehensive health history and performing a physical assessment with a focus on the musculoskeletal system. In addition, assess for extra-articular manifestations of RA. Inspect the patient's joints for edema, nodules, and deformities, and ask about pain and stiffness in the joints. Determine the patient's gait stability and hand dexterity, along with the ability to perform activities of daily living (ADLs).[18]

Maintain a safe environment free from obstacles and monitor the patient for difficulty with ambulation and fine motor or gross motor skills. Assess the patient's nutritional status, which may be decreased due to feeding difficulty and GI upset from some medications. Encourage small frequent meals with increased protein and vitamins. Verify that appropriate consults are obtained for physical therapy, occupational therapy, and nutritional intervention as needed. Monitor lab test results, including ESR, CBC, C4, CRP, and ANA results, and report any abnormalities to the patient's healthcare provider.[18]

Question the patient and family about home environmental safety, assessing for problems such as cluttered rooms, poor furniture arrangement, and throw rugs. Educate them on ways to increase the safety of the home environment; for example, by installing handrails in the bathroom. Monitor patients for mood changes and depression, which are common in patients with RA due to the chronicity of the disease and its negative impact on quality of life. Provide information about RA support groups in the area.[18]

Patient education

Patients need to understand that RA is a chronic disease that may impact their ADLs. Educate them and their families on the pathophysiology, risk factors, manifestations, available treatment options, medication risks and benefits, and potential physical limitations as the disease progresses. Teach patients to take their medications as prescribed and follow up with all scheduled healthcare provider appointments and lab testing. Tell them to report any uncontrolled pain, increased difficulty with ADLs, or fine motor skill deterioration to their healthcare provider. Because many patients with RA experience unintended weight loss, educate them about healthy eating habits. Encourage patients to report signs of depression to their healthcare provider.[12]

Improving quality of life

RA is a chronic, progressive disorder that can severely impact health and quality of life. With thorough assessment, appropriate referrals, nonpharmacologic interventions, and evidence-based pharmacotherapy, nurses can help keep patients with RA safe and physically active for as long as possible. ■

REFERENCES

1. Song GG, Bae SC, Kim JH, Lee YH. Association between TNF-α promoter -308 A/G polymorphism and rheumatoid arthritis: a meta-analysis. *Rheumatol Int*. 2014;34(4): 465-471.

2. Porth CM. *Pathophysiology. Concepts of Altered Health States*. 4th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2014.

3. Oliver S. Improving long-term outcomes for rheumatoid arthritis: evidence-based care. *Primary Health Care*. 2010;20(9):30-39.

4. Smeltzer SC, Bare BG, Hinkle JL, Cheever KH. *Brunner & Suddarth's Textbook of Medical-Surgical Nursing*. 12th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2010.

5. Walker J. Rheumatoid arthritis: role of the nurse and multidisciplinary team. *Br J Nurs*. 2012;21(6): 334-339.

6. Venables PJW, Maini RN. Clinical manifestations of rheumatoid arthritis. UpToDate. 2014. www.uptodate.com.

7. Matteson EL, Davis JM. Overview of the systemic and nonarticular manifestations of rheumatoid arthritis. UpToDate. 2015. www.uptodate.com.

8. Venables PJW, Maini RN. Diagnosis and differential diagnosis of rheumatoid arthritis. UpToDate. 2015. www.uptodate.com.

9. American College of Rheumatology. Rheumatoid arthritis. 2015. www.rheumatology.org/I-Am-A/Patient-Caregiver/Diseases-Conditions/Rheumatoid-Arthritis.

10. U.S. National Library of Medicine. Complement component 3 (C3). 2013. https://www.nlm.nih.gov/medlineplus/ency/article/003539.htm.

11. Schur PH, Moreland LW. General principles of management of rheumatoid arthritis in adults. UpToDate. 2015. www.uptodate.com.

12. Adams M, Koch R. *Pharmacology: Connections to Nursing Practice*. Upper Saddle River, NJ: Pearson; 2010.

13. Singh JA, Furst DE, Bharat A, et al. 2012 Update of the 2008 American College of Rheumatology Recommendations for the use of disease-modifying antirheumatic drugs and biologic agents in the treatment of rheumatoid arthritis. *Arthritis Care Res (Hoboken)*. 2012;64(5):625-639.

14. Boehringer-Ingelheim. Prednisone prescribing information. 2012. http://bidocs.boehringer-ingelheim.com/BIWebAccess/ViewServlet.ser?docBase=renetnt&folderPath=/Prescribing+Information/PIs/Roxane/Prednisone+Reformulated/PredniSONE Reform.pdf.

15. Sanofi-Aventis. Plaquenil prescribing information. 2014. http://products.sanofi.ca/en/plaquenil.pdf.

16. Pfizer. Azulfidine prescribing information. 2014. www.accessdata.fda.gov/drugsatfda_docs/label/2014/007073s128lbl.pdf.

17. Xanodyne Pharmacal. Methotrexate sodium for injection. 2003. www.accessdata.fda.gov/drugsatfda_docs/label/2004/11719slr106_methotrexate_lbl.pdf www.ncbi.nlm.nih.gov/pubmed/22473917.

18. Ignatavicius D, Workman M. *Medical-Surgical Nursing: Patient-Centered Collaborative Care*. 7th ed. St. Louis, MO: Elsevier; 2013.

19. Centers for Disease Control and Prevention. Rheumatoid arthritis. www.cdc.gov/arthritis/basics/rheumatoid.htm#4.

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