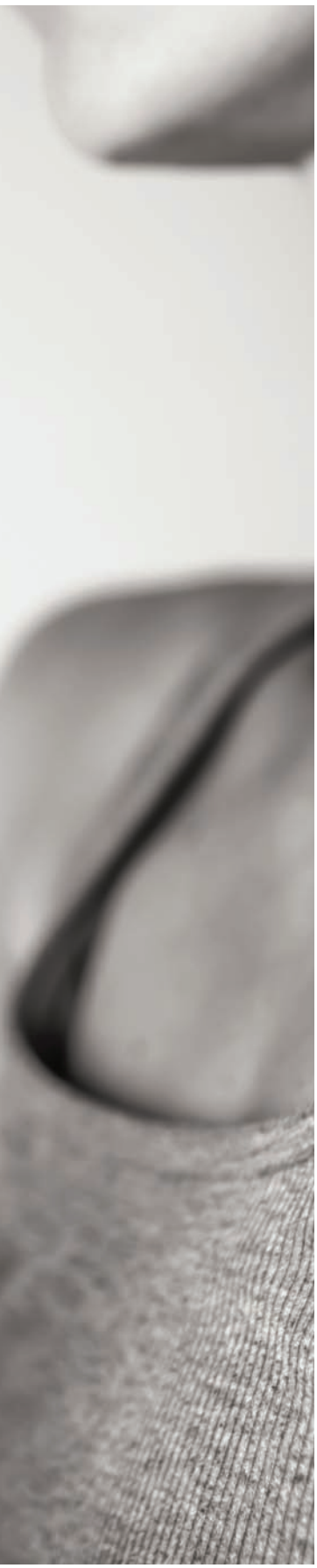

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Rheumatoid arthritis

Rheumatoid arthritis is a chronic condition that requires an interprofessional team, including the nurse, to manage the patient's overall quality of life.

By Richard L. Pullen, Jr., EdD, MSN, RN, CNE, CNE-cl, ANEF, FAAN and Lori Hammond, DNP, MSN, RN, CNE-cl, GERO-BC

Rheumatoid arthritis (RA) is an autoimmune illness caused by inflammation of the tendon and synovium (tenosynovitis), resulting in cartilage destruction, bone erosion, joint swelling, stiffness, and pain.[1,2] RA typically begins in the peripheral joints, moves proximally, is usually bilateral and symmetrical, and has multiorgan manifestations.[1,2] It's important for nurses to be aware of the pathophysiology, symptoms, and treatment of RA, because progressive joint destruction leads to irreversible joint deformity, chronic pain, and disability.[1,2] This article discusses the pathophysiology, signs, symptoms, and management of patients with RA from an interprofessional perspective. Nurses practice alongside many colleagues, including but not limited to physicians, pharmacists, physical therapists, and occupational therapists to help the patient with RA to have a better quality of life.

Epidemiology and risk factors

RA is a significant health problem affecting 0.5% to 1% of all populations globally, including more than 1.3 million people in the US. A 75% of patients with RA are female.[3-5] The onset of RA can happen at any age, but it usually begins between ages 30 and 50.[3-5] There's an increased risk of developing RA in people whose first-degree relatives have the disease.[3-5] A five-sequence chain of the human leukocyte antigen DRB1, known as a shared epitope, is the primary genetic influence in the development of RA.[5,6]

The gut plays a significant role in immune function.[7] Imbalances in gut microbiota can lead to autoimmune illnesses, including RA.[7] For instance, excessive consumption of certain foods is a risk factor for RA because they cause inflammation. A high body mass index is also a risk factor, and correlates with increased inflammation.[5,8,9] Patients should avoid smoking because it has an inflammatory effect and exacerbates inflammation by circulating autoantibodies associated with RA.[5] Periodontal disease, dust, pollen, and infections are also risk factors for RA.[5]

Pathophysiology

RA is an immune-mediated illness, meaning a person's immune system becomes overactive and doesn't recognize their healthy body tissues as belonging to them.[10] The overactive immune system causes inflammation and tissue damage, initially in the synovium, the thin

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protective lining of a joint known as the tendon sheath that provides nutrients and blood supply to cartilage.[10] Collagen enables the cartilage to support the joint, while synovial fluid lubricates the cartilage. Degradation of the synovium is triggered by environmental factors in a genetically susceptible person (in this case, a person with RA) and involves complex immunologic activation and inflammatory pathway processes.[10]

Macrophages are normally present in the synovium.[10-13] In RA, macrophages secrete proinflammatory cytokines such as tumor necrosis factor-alpha and interleukin-a, and stimulate the production of fibroblast-like synoviocytes (FLS), causing inflammation.[10-13] Fibroblasts are specific cells for collagen development, whereas FLSs are nonimmune cells essential for developing the synovial lining. FLSs stimulate the production of proinflammatory cytokines and the receptor activator of nuclear factor-kappa B ligand (RANKL), a protein on the surface of osteoclasts that stimulates osteoclastic activity, causing bone erosion.[10-13]

When FLS levels increase, they secrete enzymes that contribute to cartilage degradation and migrate symmetrically from one joint to another.[10-13] T lymphocytes are integral components of synovium that promote inflammation and secrete interleukin-17 to stimulate macrophage and FLS activity and the development of RANKL.[10-13] Synovial fluid contains neutrophils, which produce enzymes and reactive oxygen species, an unstable molecule that damages cells and leads to apoptosis, causing cartilage and bone degradation.[10-13] Synovial fluid contains immune complexes (one antigen + one antibody together). Angiogenesis increases the vascular permeability of cells to enable immune complexes to migrate to joints.[10-13]

Researchers postulate factors such as genetics, smoking, and infections can trigger an alteration in autoantigens, which become foreign to a person's immune cells.[10] Alteration in autoantibodies is called citrullination and results in synovial injury, joint infection, and joint hyperplasia.[10] Joint inflammation stimulates more proinflammatory cytokines to alter autoantigens further.[10] Antigenic cells recognize the altered autoantigens to initiate an immune response. The antigenic cells migrate to lymph nodes and activate CD4 T lymphocytes and B lymphocytes. B lymphocytes proliferate, produce autoantibodies against a person's antigens, and migrate to joint tissue.[10]

Two autoantibodies commonly identified in patients with RA, anticitrullinated protein antibody (ACPA) and rheumatoid factor (RF), are used to diagnose RA.[2,14] RF occurs in most patients with RA, but it also occurs in other autoimmune illnesses, including but not limited to systemic lupus erythematosus, Sjögren syndrome, scleroderma, and juvenile idiopathic arthritis. ACPA is highly specific for RA.[2,14] Multiorgan involvement results from the proinflammatory cytokines produced within the joints and

correlates with a poor outcome.[1,2,15] New evidence indicates that inflammatory processes leading to RA may originate from immune dysregulation in the respiratory, digestive, and reproductive tract's mucosal lining.[10] Patients in this preclinical scenario who are positive for RF and ACPA have elevated inflammatory markers such as c-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), and have no joint symptoms.[2,14] Some people with RA don't possess RF and ACPA, but they have other features of the illness.This is known as seronegative RA.[2,14,16]

Signs, symptoms, and diagnostics

RA usually starts in the joints of the feet, hands, and wrists, and progresses to the ankles, knees, hips, elbows, cervical spine, and temporomandibular joints.[1,2,7] Patients experience stiffness and swelling lasting more than 1 hour after arising from a period of rest, which is suggestive of an inflammatory process.[1,2,7] Early in the disease, joint stiffness may improve with activity and gradually worsen over time. Severe RA may result in permanent destruction of the cartilage and joint. Patients may experience joint dislocation.

Patients may experience low-grade fever, fatigue, anorexia, and weight loss.[1,2,7] The presence of RF, ACPA, and antinuclear antibodies indicates the immune-mediated nature of the illness.[2] Increased CRP and ESR are reliable inflammatory markers of disease activity, and are used to monitor medication effectiveness.[2] A complete blood cell count often reflects anemia secondary to inflammation and correlates with the severity of fatigue that a patient may experience.[2] Reduced white blood cells and platelets may occur secondary to medication therapy, predisposing the patient to infection and bleeding.[2] Elevated liver enzymes and creatinine may indicate toxicity from medication therapy.[2] X-rays are the standard imaging modality to identify the narrowing of joint spaces and bone erosion.[2,17] Computed tomography and ultrasounds may also be used.[2,17] MRI has the added benefit of showing soft tissue and synovial involvement.[17] An analysis of synovial fluid by needle aspiration often reveals proinflammatory cytokines, immune complexes, macrophages, and FLSs, which are diagnostic of RA.[2,11] (See *1987 classification for RA*, *2010 classification for RA*, and *Selected extra-articular manifestations of RA*.)

1987 classification for RA

Adapted from the 1987 American College of Rheumatology Classification Criteria. Four or more are required for a diagnosis of RA.[2]

- Morning joint stiffness lasting more than 1 hour
- Arthritis with joint swelling in three or more joints
- Arthritis of the hand joints
- Symmetric arthritis
- Rheumatoid nodules
- Rheumatoid factor positivity
- Radiographic changes

2010 classification for RA

Revised criteria were created by the American College of Rheumatology and the European League Against Rheumatism in 2010 to identify patients with early RA and prevent illness progression and may be used in conjunction with the 1987 criteria. A total of six points or greater is required for an RA diagnosis.[2]

Number of joints and point allocation	
• 1 large joint	0
• 2 to 10 large joints	1
• 1 to 3 small joints	2
• 4 to 10 small joints	3
• More than 10 joints plus 1 small joint	5
Autoantibodies and point allocation	
• Negative RF and ACPA	0
• Low-positive RF or ACPA	2
• High-positive RF or ACPA	3
CRP and ESR and point allocation	
• Normal CRP and ESR	0
• Elevated CRP or ESR	1
Persistence of symptoms and point allocation	
• Less than 6 weeks	0
• 6 weeks or longer	1

Selected extra-articular manifestations of RA[1,2,15]

Body system	Manifestation
Integumentary	• Rheumatoid nodules • Cutaneous vasculitis • Palmar erythema
Pulmonary	• Pleuritis • Interstitial lung disease • Pneumonia
Cardiovascular	• Accelerated atherosclerosis • Hypertension • Conduction disorders • Myocardial infarction • Heart failure • Pericarditis
Central and peripheral nervous systems	• Cerebral vasculitis • Neuropathy • Cerebrovascular accident • Reduced cognition
Hematologic	• Anemia • Thrombocytopenia • Neutropenia
Immune	• Splenic involvement: Infections
Musculoskeletal	• Osteoporosis
Ocular	• Scleritis • Keratoconjunctivitis

Management

Pharmacologic treatment is a standard intervention when a patient experiences an RA flare.[18-20] Pharmacologic treatment aims to reduce inflammation and place the patient in remission, achieved when symptoms are controlled, and the patient has a quality of life.[18-20] Medication classifications to treat RA include nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, disease-modifying antirheumatic drugs (DMARDs), small-molecule Janus kinase inhibitors, and biological response modifiers (BRMs).[18-25] NSAIDs effectively reduce inflammation and pain but should be used at the lowest therapeutic dose and for a short period to avoid gastrointestinal (GI), renal, and cardiovascular complications.[21] Acetaminophen may be used to reduce pain, but it doesn't have anti-inflammatory properties.[21] Opioids may be explored by the pain management team when patients haven't achieved relief from NSAIDS and nonpharmacologic treatment strategies.[21] Systemic corticosteroids quickly reduce inflammation, pain, and progression of RA. Still, they should be prescribed in the lowest possible dose and for a short period to limit severe cumulative adverse reactions, including but not limited to changes in mental status, sodium retention, weight gain, problems regulating blood glucose, and hypertension.[18]

Intra-articular injections of corticosteroids should be considered to reduce inflammation, swelling, and pain.[18] DMARDs reduce systemic inflammation to improve symptoms.[18,22] BRMs target inflammatory cells and pathways to slow joint degeneration.[23-25] DMARDs, BRMs, and corticosteroids cause immunosuppression, which predisposes patients to infection.[18,22-25] (See *Examples of medications in RA and priority assessments*.)

Pharmacologic interventions complement nonpharmacologic strategies and include physical therapy, hydrotherapy, massage, arthroscopy to repair damaged joints and remove excessive fluid, joint replacement surgery, orthotic devices, splints, exercise as tolerated, balance exercises to prevent falls, diet and weight control, and pain management. Early diagnosis and aggressive treatment will help to reduce joint damage and disability.

Nursing interventions

Chronic illnesses, such as RA, require an interprofessional team (IPT) to manage the patient's physical, psychological, and spiritual sources of suffering. The IPT may include nurses, physicians, pharmacists, physical therapists, occupational therapists, dietitians, counselors, psychologists, pastoral care, and social workers. Nurses must conduct a health history,

Examples of medications in RA and priority assessments

Medication	Priority assessments
NSAIDs • Ibuprofen • Diclofenac • Naproxen • Piroxicam • Celecoxib • Meloxicam **Therapeutic action:** Inhibition of cyclooxygenase-associated prostaglandin production to decrease inflammation and pain.	Should be taken with food or water. • Joint inflammation, swelling, pain, and mobility. • Thrombosis: Medication accelerates atherosclerosis. • Signs and symptoms of bruising or bleeding. • Bright red blood in stool or black, tarry stools. • Bright red or coffee-ground color emesis. • Reduced platelet (PLT) and prolonged partial thromboplastin time (PTT): Bleeding. • Elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP): Hepatic toxicity. • Elevated creatinine and blood urea nitrogenc: Renal toxicity. • Visual acuity and hearing ability.
Corticosteroids • Prednisone • Prednisolone • Methylprednisolone • Dexamethasone • Hydrocortisone • Triamcinolone • Budesonide **Therapeutic action:** Suppresses migration of leukocytes and reversal of increased capillary permeability to decrease inflammation and causes immunosuppression.	Corticosteroids alter fluid and electrolytes and the metabolism of carbohydrates, proteins, and fats and influence neurologic, cardiovascular, immunologic, endocrine, and musculoskeletal balance. • Joint inflammation, swelling, and pain. • Mood changes: Medication alters neurotransmitters in the brain. • Hypertension. • Dysrhythmias. • Bleeding. • Capillary fragility with easy bruising. • Hypokalemia and hypernatremia. • Weight gain: Increased appetite, sodium retention, edema. • Increased blood glucose, ALT, AST, ALP, and cholesterol. • Reduced PLTs and prolonged PTT. • Medication masks infection: Potential for severe infection. • Fractures: Osteoporosis. • Visual changes: Cataracts.
DMARD • Methotrexate **Therapeutic action:** Inhibits the production of pro-inflammatory cytokines to reduce inflammation in RA. Immunosuppression. Inhibits purine and pyrimidine synthesis affecting mitosis in malignancy.	• Joint inflammation, swelling, and pain. • Elevated ALT, AST, and ALP: Hepatic toxicity. • Reduced PLTs and prolonged PTT: Bleeding. • Elevated creatinine: Renal toxicity. • Neutropenia: Potential for severe infection. • Fluid and electrolyte imbalances. • Medication reduces folic acid for red blood cell (RBC) production. • Decreased RBC production indicating anemia. • Photosensitivity: Wear SPF of ≥30. • Oral ulcerations.
DMARD • Hydroxychloroquine **Therapeutic action:** Inhibits proinflammatory cytokine release, slows movement of neutrophils, and impairs antibody-antigen responses to reduce inflammation in RA. Mild immunosuppression. Used for malaria by blocking the detoxification of plasmodium parasites and the lysosomal degradation of hemoglobin.	• Joint inflammation, swelling, and pain. • Elevated ALT, AST, and ALP: Hepatic toxicity. • Elevated creatinine: Renal toxicity. • Neutropenia: Infection. • Dysrhythmias: Baseline and periodic ECG. • Visual problems: Retinal damage (reversible). • Bleeding, bruising, and skin eruptions. • Hypoglycemia: Medication increases insulin.

(continues)

Examples of medications in RA and priority assessments *(continued)*

Medication	Priority assessments
DMARD • Sulfasalazine **Therapeutic action:** Induces T lymphocytes apoptosis, inhibits activation of B lymphocytes, inhibits production of prostaglandins, scavenges toxic oxygen metabolites to reduce inflammation in RA, and has some antibacterial action. Mild immunosuppression.	Medication should be taken after eating to avoid GI upset. • Joint inflammation, swelling, and pain. • Periodic RBC and folate levels: Anemia. • Elevated ALT, AST, and ALP: Hepatic toxicity. • Elevated creatinine: Renal toxicity. • Neutropenia: Infection. • Headache, anorexia, nausea, vomiting, diarrhea. • Reduction in sperm count.
DMARD • Leflunomide **Therapeutic action:** Suppresses synthesis of T lymphocytes and B lymphocytes and interferes with protein and RNA cells involved in inflammation of joints in RA. Immunosuppression.	• Joint inflammation, swelling, and pain. • Elevated ALT, AST, and ALP: Hepatic toxicity. • Neutropenia: Potential for severe infection. • Reduced PLTs and prolonged PTT: Bleeding. • Hypertension, headache, nausea, diarrhea, and alopecia. • Neuropathy and paresthesia.
Small-molecule DMARD • Tofacitinib **Therapeutic action:** Suppresses multiple Janus kinases signal transducer and activator of transcription of protein pathways in proinflammatory cytokines to reduce inflammation in RA.	• Joint inflammation, swelling, and pain. • Lymphocytosis (RA onset) followed by neutropenia. • Potential for severe infection. • Increased risk for malignancy. • Elevated ALT, AST, and ALP: Hepatic toxicity. • Elevated creatinine: Renal toxicity. • Reduction in RBCs: Anemia. • Increased cholesterol. • Fatigue, headache, insomnia, nasopharyngitis. • Nausea, vomiting, diarrhea. • Fluid and electrolyte imbalances.
BRMs **Therapeutic action:** Monoclonal antibodies target specific proteins to reduce inflammation in RA. **Tumor necrosis factor inhibitors:** • Adalimumab • Infliximab **Interleukin-6 inhibitor:** • Tocilizumab **Anti-CD-20 monoclonal antibody:** • Rituximab	• Joint inflammation, swelling, and pain. • Neutropenia. • Potential for severe infection. • Increased risk for malignancy. • Elevated ALT, AST, and ALP: Hepatic toxicity. • Reduced RBCs: Anemia. • Reduced PLTs and prolonged PTT: Bleeding. • Fatigue, anorexia, nausea, vomiting, diarrhea. • Skin rashes and potential erythema multiforme.

perform a head-to-toe assessment, evaluate comorbidities, evaluate the severity of pain and disability, correlate lab and imaging studies reflecting inflammation, and evaluate the patient's emotional status. The IPT, including the nurse, will assess the patient's responses to pharmacologic and nonpharmacologic treatment strategies. Managing the following will promote quality of life.[1,4,9,18,20,26-34]

1. **Severe pain**. Support warm, swollen, and tender joints with pillows, apply warm compresses to relax muscles and stiff joints, and apply cool compresses to reduce swelling. Administer analgesics as needed. A pain management team is often necessary. Systemic inflammation causes pain, fatigue, anemia, and elevated CRP and ESR. Chronic inflammation depletes erythropoietin, a hormone that stimulates RBC production, causing anemia.
2. **Medication responses**. Systemic medications are often necessary to control inflammation. The efficacy of these medications is measured with an improvement of symptoms, a reduction in CRP and ESR, a decrease in RF and ACPA, and minimal to no adverse reactions. Nurses should teach patients to report adverse reactions of medications, including but not limited to an increase in joint redness, swelling, pain, the onset of fever, bleeding from anywhere in the body, weight gain or loss, changes in mental status, abdominal pain or discomfort, and skin rashes and lesions, especially concerning in BRMs (see *Examples of medications in RA and priority assessments*).
3. **Nutrition and weight**. Foods with anti-inflammatory properties should be encouraged, including fish, vegetables, fruits, beans, legumes, seeds, whole grain bread, oats, olive oil, tofu, lean turkey, and chicken breasts. These foods are rich in iron to help with anemia. Saturated fats should be avoided, including processed food, fast food, fried food, baked goods, and sugar. Being overweight and obese causes pressure on painful joints and adipocytes (fat cells) to release proinflammatory cytokines which worsen RA. A well-balanced diet, sufficient hydration, and vitamin supplements (such as a multivitamin) combined with physical activity promote an optimal BMR. Patients should consult a dietitian to help them with healthy food choices.
4. **Exercise**. Patients should engage in range of motion, flexibility, and strengthening exercises as tolerated to loosen stiff joints and relax muscles. Walking, riding a stationary bike, and wading in a swimming pool are helpful. Advise patients to slowly integrate low-impact exercises to prevent additional joint injury, increase mobility, and decrease pain over time.
5. **Comorbidities**. Common comorbidities related to inflammation in RA, complications of medications, or coping with chronic illness are infections, GI, renal, cardiovascular and pulmonary disorders, osteoporosis, malignancy, and depression. Patients may have comorbidities unrelated to RA before the onset of symptoms. Smoking, excessive consumption of alcohol, and a lack of physical activity can exacerbate symptoms of comorbidities.
6. **Assistive devices**. Assistive devices can help compensate for physical limitations in RA, including: (1) utensils with large handles made out of foam or rubber for an easier grip, (2) shoes with special inserts or orthopedic shoes for support, (3) special devices for dressing, (4) bathtub chair lifts and shower stools, and (5) combs, brushes, and sponges with long handles.
7. **Self-care**. Mindfulness is an intentional process where people meditate about their thoughts, feelings, and environment. Self-care includes, but isn't limited to: (1) adhering to the treatment plan by the IPT, (2) receiving vaccinations as indicated, (3) avoiding people who are sick, (4) wearing an N95 or KN95 mask if receiving immunosuppressive medications, (5) keeping appointments with healthcare providers, (6) getting plenty of sleep, (7) resting at intervals, (8) being physically active, (9) getting a hobby, and (10) engaging in support groups (see *On the web*).

On the web

Selected resources for nurses, patients, and families

Resource	Primary focus
American College of Rheumatology: www.rheumatology.org/I-Am-A/Patient-Caregiver/Diseases-Conditions/Rheumatoid-Arthritis	Education resources for patients and caregivers about the various types of arthritis.
Centers for Disease Control and Prevention: www.cdc.gov/arthritis/healthcare/index.html	Care for patients with arthritis. Patient-care guidelines and resources for patients, families, and healthcare providers.
Arthritis Foundation: www.arthritis.org/	Different types of arthritis, treatment strategies, and lifestyle modification to promote quality of life.
Rheumatology Research Foundation: www.rheumresearch.org/	Teaching about RA, research about RA, and patients sharing their experiences with RA.

Putting it all together

RA is a complex illness requiring a nurse to use critical thinking and clinical reasoning to manage the patient safely. RA is a chronic, debilitating, inflammatory illness affecting joints, body organs, and tissues with varying severity among patients. Early diagnosis and treatment will help prevent severe joint damage, functional impairment, and pain. Pharmacologic, nonpharmacologic, and self-care strategies will promote quality of life. ■

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