

Rheumatoid Arthritis

A plain-English guide for you and your family

WHAT IS HAPPENING

Healthy joints have a soft lining called the synovium that makes slippery fluid so bones can glide smoothly. Your immune system normally ignores your own body parts and only fights germs. In rheumatoid arthritis, that changes. Your immune system starts making proteins called autoantibodies (antibodies that attack your own tissue) that flag the synovium as dangerous. White blood cells flood into the joint, release chemicals that cause swelling and pain, and the synovium grows thick and angry. Over time, this thick lining makes enzymes (chemicals that break down tissue) that eat away the cartilage cushion and even the bone underneath. Because the immune attack travels through your bloodstream, it usually hits matching joints on both sides of your body at the same time—both wrists, both sets of knuckles. You wake up with stiff, swollen fingers that take more than an hour to loosen up. You feel tired and may run a low fever because your whole body is inflamed. If the attack keeps going for months or years without treatment, the bones and tendons change shape and your fingers can bend sideways or curve into a swan-neck. The inflammation can spread beyond joints to your lungs, heart, blood vessels, eyes, and skin. None of this is random—every symptom traces back to one upstream break: your immune system fighting your own joint lining.

What you might feel

- Small joints in the hands and feet are swollen, warm, and tender on both sides of the body
- Morning stiffness that lasts over an hour
- You feel very tired all the time
- The DIP joints (the fingertip knuckles) are NOT swollen or sore
- Rheumatoid nodules (firm lumps under the skin, often near elbows)
- Low-grade fever
- Joint damage or deformity (in advanced or untreated disease)
- Lung problems (shortness of breath, dry cough) or chest pain

Call the doctor right away if...

- Severe joint pain that suddenly gets much worse — may mean infection in the joint (septic arthritis) on top of rheumatoid arthritis
- New shortness of breath, persistent dry cough, or chest pain — may mean lung involvement (pleuritis or interstitial lung disease)
- Fever above 101°F (38.3°C) — may mean infection, especially if on immune-suppressing medicines
- Sudden vision changes or severe eye pain — may mean scleritis (inflammation of the white of the eye), which can damage vision
- Visible joint deformity or inability to use the hand or foot — means the disease is advancing and needs urgent treatment adjustment

What the care team will do

- Check both hands and feet for symmetrical joint swelling, warmth, and tenderness, and document which specific joints are affected
- Assess cervical spine stability by asking the patient to gently tuck chin to chest and report any electric-shock feelings down the spine or new arm weakness
- Review the medication list to confirm the patient takes folic acid (1 mg daily) on the days they do not take methotrexate
- Teach proper joint protection techniques like using larger joints to carry loads, avoiding tight grip positions, and using assistive devices for jars and buttons
- Monitor for signs of infection including fever over 100.4 degrees Fahrenheit, new cough, burning with urination, or a single hot swollen joint
- Educate the patient that NSAIDs like ibuprofen only mask pain but do not stop joint damage, while DMARDs and biologics actually slow disease progression
- Screen for depression and offer referrals to occupational therapy and support groups
- Schedule baseline tuberculosis testing (QuantiFERON or PPD skin test) and hepatitis B screening before starting biologic therapy

Medicines you may be given — and why

Methotrexate

Methotrexate blocks an enzyme called dihydrofolate reductase, which immune cells need to copy their DNA and multiply. When the enzyme is blocked, the fast-growing immune cells that attack the joints slow down and make fewer inflammatory signals like TNF-alpha and interleukin-1.

Hydroxychloroquine

Hydroxychloroquine gets concentrated inside immune cells and raises the pH (makes the inside less acidic) of little sacs called lysosomes and endosomes. This change makes it harder for immune cells to process antigens (foreign or self pieces) and activate, so fewer inflammatory signals are sent out.

Sulfasalazine

Sulfasalazine is broken down in the gut into sulfapyridine and 5-aminosalicylic acid. The sulfapyridine part is thought to lower inflammation by blocking enzymes (like cyclooxygenase and lipoxygenase) that make inflammatory prostaglandins and leukotrienes, and by reducing immune cell activity.

Adalimumab, Etanercept, Infliximab, Golimumab, Certolizumab

These drugs grab onto TNF-alpha (a powerful inflammatory messenger made by immune cells) in the blood and joints before it can lock onto receptors on other cells. By blocking TNF-alpha, the chain reaction of inflammation — more immune cells swarming in, more enzymes chewing up cartilage and bone — is interrupted.

Abatacept

T cells (a type of white blood cell that directs the immune attack) need two signals to fully activate: one from recognizing an antigen and one from a helper molecule called CD28. Abatacept blocks the second signal by attaching to CD80 and CD86 (proteins on antigen-presenting cells that normally give the go-ahead). Without both signals, T cells stay quiet and do not call in more inflammation.

Tocilizumab, Sarilumab

Interleukin-6 (IL-6) is a cytokine that tells the liver to make acute-phase proteins (like CRP), tells bone cells to break down bone, and keeps inflammation going. Tocilizumab and sarilumab block the IL-6 receptor on cells, so IL-6 cannot deliver its inflammation signals.

Rituximab

B cells are white blood cells that make antibodies, including the autoantibodies (like rheumatoid factor and anti-CCP) that attack the joints in rheumatoid arthritis. Rituximab is an antibody that locks onto CD20, a protein on the surface of B cells, marking them for destruction by the immune system. Wiping out B cells lowers autoantibody levels and calms the immune attack.

Tofacitinib, Baricitinib, Upadacitinib

Many inflammatory cytokines (like IL-6, interferon, and others) send their signals into cells by activating enzymes called Janus kinases (JAK1, JAK2, JAK3, TYK2). JAK inhibitors block these enzymes, so the cytokine signals cannot turn on the genes that keep inflammation going. This dials down the immune attack on joints.

Prednisone, Methylprednisolone

Steroids enter cells and bind to receptors in the cytoplasm. The steroid-receptor pair moves into the nucleus and changes which genes are turned on or off. This lowers the production of many inflammatory chemicals (cytokines, prostaglandins, leukotrienes) and tells immune cells to calm down. The effect is fast and powerful.

Ibuprofen, Naproxen, Celecoxib, Diclofenac

NSAIDs block enzymes called cyclooxygenase-1 and cyclooxygenase-2 (COX-1 and COX-2), which make prostaglandins. Prostaglandins are chemicals that cause pain, fever, swelling, and (in the stomach) protect the lining. Blocking them lowers pain and inflammation but also raises the risk of stomach ulcers and bleeding.

Where this information comes from

- American College of Rheumatology (ACR) 2021 Guideline for the Treatment of Rheumatoid Arthritis
- European League Against Rheumatism (EULAR) 2022 Recommendations
- UpToDate Clinical Overview of Rheumatoid Arthritis
- Cochrane Review: DMARDs for Rheumatoid Arthritis

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