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A Comprehensive Review of Pathogenesis, Symptoms, and Therapeutic Approaches in Rheumatoid Arthritis

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Abstract: *The systemic autoimmune illness known as rheumatoid arthritis (RA) is typified by extra-articular involvement and inflammatory arthritis. Mostly affecting synovial joints, it is a chronic inflammatory disease that is frequently brought on by the interplay of genes and environmental factors, such as tobacco [1,2]. It usually begins in tiny peripheral joints, is symmetrical, and, if treatment is not received, spreads to proximal joints [3]. Inflammation of the joint causes bone erosion and cartilage loss over time, ultimately resulting in joint disintegration [4]. Early RA is characterized by symptoms that appear within six months, while established RA is characterized by symptoms that have persisted for longer than six months [5]. If left untreated, RA worsens over time and increases morbidity and mortality [3,6].*

Keywords: Rheumatoid Arthritis disease, Diagnosis, Treatment, Drug

I. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic systemic inflammatory disease that primarily affects the elderly and is more common in women than in men [7]. The 2002 prevalence rate varied by region and ranged from 0.5% to 1% of the population [1,8]. The lining of the synovial joints is the main target of RA, which can lead to progressive disability, early death, and financial hardships [3,9]. Arthralgia, edema, redness, and a restriction in range of motion are clinical signs of symmetrical joint involvement [7]. For the best results—less joint destruction, radiologic progression, and disability—early diagnosis and disease-modifying antirheumatic drug (DMARD)-free remission are essential [8,9].

The Ideal therapeutic window is thought to be within weeks after the onset of early symptoms [10]. Early diagnosis is still difficult, as it depends on medical history, physical examination, serological testing, and imaging [11]. Delays in DMARD initiation may depend on patient awareness, access to care, and physician diagnosis [8,12].

History

The history of rheumatoid arthritis dates back hundreds of years [13]. The disease was first clearly described in 1800 by French physician Augustin Jacob Landré-Beauvais, who noted its differences from gout and osteoarthritis [6]. He observed that the disease affected mostly women and people of lower socioeconomic status [6,13]. In 1890, the term “rheumatoid arthritis” was introduced [13].

Early therapies included hot-water baths and cold compresses [13]. The introduction of glucocorticoids in 1948 revolutionized symptom control [14], and later, DMARDs such as methotrexate and sulfasalazine helped slow disease progression [15].

Drugs Used in the Treatment of Rheumatoid Arthritis

1. NSAIDs – Used to reduce pain and inflammation only [16].
Examples: Ibuprofen, Diclofenac.
2. Corticosteroids – Provide rapid relief of inflammation; used short-term [17].

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Examples: Prednisolone, Dexamethasone.

3. MARDs (Disease-Modifying Anti-Rheumatic Drugs) – Slow disease progression and prevent joint damage [8],[15].
4. Conventional: Methotrexate, Sulfasalazine, Hydroxychloroquine
5. Biological: Etanercept, Infliximab, Adalimumab
6. Targeted synthetic DMARDs – Tofacitinib, Baricitinib [9].
7. Adjunct therapy – Supportive treatments like analgesics, calcium, vitamin D [16].

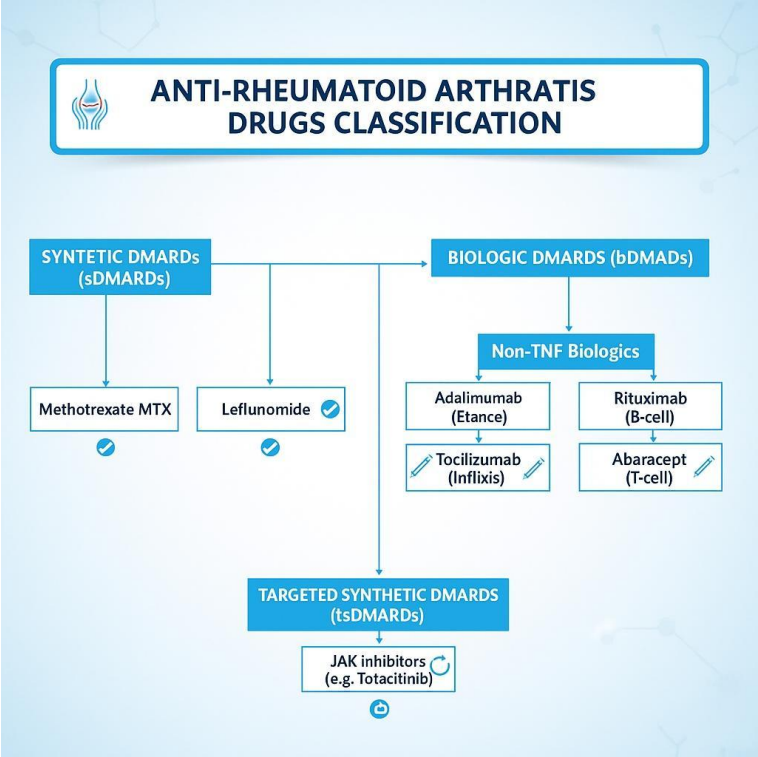

Fig. No. 1-Classification of Anti-Rheumatoid Drug

Symptoms of Rheumatoid Arthritis

1. Joint pain and swelling – Especially in small joints (hands, wrists, feet) [3].
2. Morning stiffness – Joints feel stiff for more than 30 minutes after waking [9].
3. Warmth and redness – Affected joints may feel warm and appear reddish [7].
4. Fatigue and weakness – Feeling tired and low in energy [10].
5. Loss of joint function – Difficulty in moving joints or doing daily activities [3].
6. Joint deformity – Long-term cases may result in visible deformities [9].
7. Systemic symptoms – Fever, weight loss, or anaemia in severe cases [3],[9].

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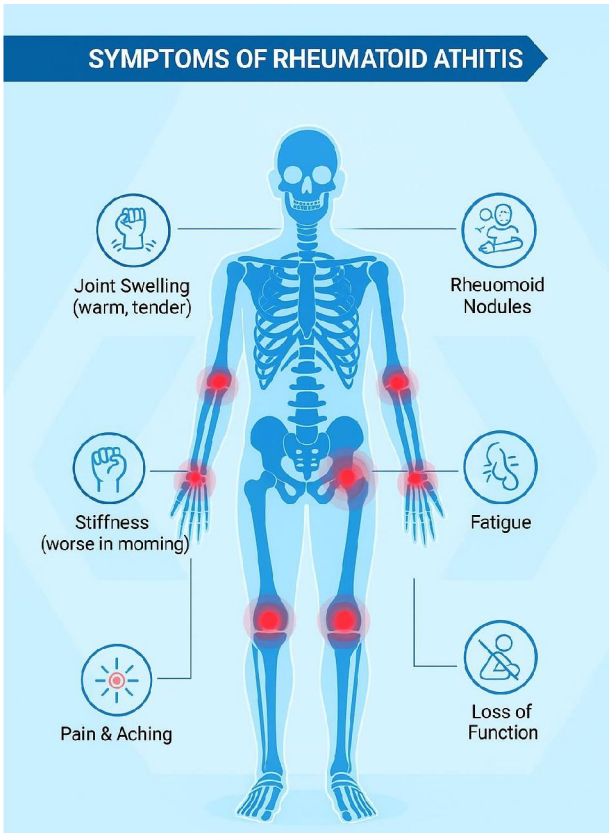

Fig. No. 2 – Symptoms of Rheumatoid Arthritis

Mechanism of Rheumatoid Arthritis

Rheumatoid arthritis (RA) is a chronic autoimmune disease where the body's immune system attacks the synovial membrane of joints [7]. Genetic predisposition (e.g., HLA-DR4) and environmental factors like smoking trigger loss of immune tolerance, activating T and B cells [7,10,11]. These cells release autoantibodies (rheumatoid factor and anti-CCP) and cytokines (TNF-α, IL-1, IL-6) that promote inflammation [7,8]. The inflamed synovium forms a pannus, which invades and destroys cartilage and bone [3,7,11].

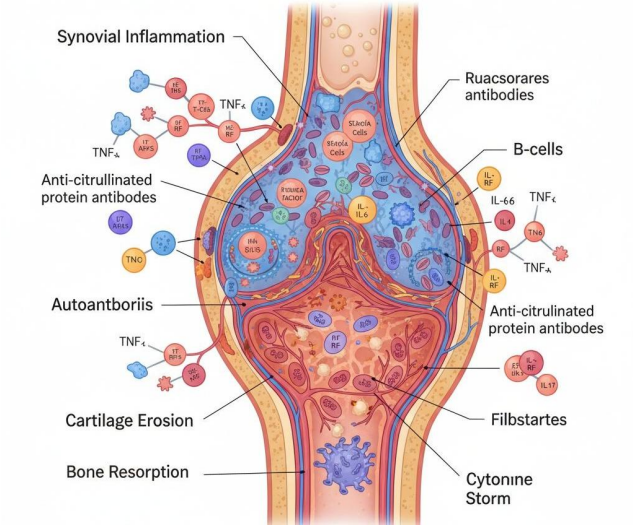

Fig. No. 3 – Mechanism of Rheumatoid Arthritis.

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Causes

Although the exact cause is unknown, RA arises from both genetic and environmental factors [7,14,19].
Genetic Factors: Variants in HLA-DRB1 genes increase susceptibility [1,3].
Environmental Factors: Smoking, infections, obesity, and periodontal disease contribute to risk [7,14,19].
Hormonal Factors: Estrogen fluctuations may influence disease onset [19].

Diagnosis

Early RA diagnosis is challenging because symptoms mimic other arthropathies [11,18].
Physical Examination: Check for joint swelling, tenderness, and range of motion [18].
Blood Tests: Elevated ESR and CRP, rheumatoid factor, and anti-CCP antibodies [18].
Imaging: X-ray, ultrasound, and MRI detect early erosions and synovial inflammation [11].

Treatment

There is no known cure for RA [3,8]. However, early DMARD therapy reduces joint damage and improves outcomes [8,9].
Regular monitoring is required to evaluate disease activity and drug toxicity [8].

Medication:

NSAIDs for pain relief [16], DMARDs (e.g., methotrexate, sulfasalazine) to slow disease progression [15], and biologics (TNF-α inhibitors such as adalimumab and etanercept) for refractory cases [8,9,16].
Targeted synthetic DMARDs (JAK inhibitors like tofacitinib and baricitinib) may be used in non-responders [9].

Therapy:

Physical and occupational therapy help maintain joint function and mobility [17].
Assistive devices reduce joint strain [17].

Surgery:

Joint replacement or repair may be required in advanced cases [8,9].

Side Effects of Anti-Rheumatoid Drugs

Table no. 1 Side Effects of Anti-Rheumatoid Drug

Drug Type	Common Adverse Effects	References
NSAIDs	Gastritis, peptic ulcers, gastrointestinal bleeding, renal toxicity	16
Corticosteroids	Weight gain, osteoporosis, hyperglycemia, mood swings	17
Conventional DMARDs	Hepatotoxicity, nausea, cytopenia, rash, ocular toxicity	15
Biologic DMARDs	Injection site reactions, increased risk of infections, rare autoimmune reactions	8,9
JAK Inhibitors (Targeted Synthetic DMARDs)	Increased infection risk, venous thromboembolism (blood clots), elevated cholesterol levels	9

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II. CONCLUSION

Rheumatoid arthritis (RA) is a chronic autoimmune disease affecting joints and multiple organs [3,7]. Although its cause is multifactorial, genetic predisposition and environmental triggers are key contributors [1,7,19]. Early diagnosis and timely initiation of DMARD or biologic therapy are crucial for preventing joint damage and improving quality of life [8,9,16]. Lifestyle changes, physiotherapy, and patient education also play important roles in long-term management [17,20].

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