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A Review on Herbal Drugs Used In the Management of Rheumatoid Arthritis

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ABSTRACT:
Pain, stiffness, swelling, and deformity are the main symptoms of rheumatoid arthritis (RA). RA is a chronic, progressive autoimmune disease that primarily affects synovial joints. Although they relieve symptoms, current conventional therapies like disease-modifying anti-rheumatic medications, steroids, and biological products are frequently linked to serious side effects, high costs, and limited long-term efficacy. Since ancient times, herbal remedies have been used to treat inflammatory conditions in Ayurveda, Traditional Chinese Medicine (TCM), and folk medicine. Strong anti-inflammatory, antioxidant, and immunomodulatory qualities are found in plants like Boswellia serrata (Indian frankincense), Tinospora cordifolia (giloy), Withaniasomnifera (ashwagandha), Zingiber officinale (ginger), and Allium sativum (garlic). In this article discusses the phytochemical components, pharmacological actions, commercial formulations, and potential in contemporary drug discovery of medicinal plants, emphasizing their role in the treatment of RA.
Some widely studied herbs in RA management include Curcuma longa (curcumin), Boswellia serrata (boswellic acids), Tinospora cordifolia (guduchi), Zingiber officinale (ginger), and Withaniasomnifera (ashwagandha,Camellia sinensis (green tea polyphenols), and Glycyrrhiza glabra (licorice). Clinical and preclinical studies suggest that these herbal formulations not only alleviate pain and swelling but also improve mobility, reduce dependence on synthetic drugs, and enhance quality of life with fewer adverse effects. Moreover, the synergistic use of herbal drugs with conventional therapy may reduce drug dosage requirements, thereby minimizing toxicity.
Despite promising pharmacological evidence, challenges remain in standardization, bioavailability, quality control, safety profiling, and large-scale clinical validation of herbal medicines. Further research in pharmacognosy, phytochemistry, and clinical trials is necessary to create evidence-based recommendations for their therapeutic application in the treatment of RA.

I. INTRODUCTION:

Rheumatoid arthritis is a long-term, progressive, systemic autoimmune disease that causes chronic inflammation mainly in synovial joints, cartilage destructionalong with bone erosion. It is characterized by pain, swelling, morning stiffness, and restricted mobility, eventually resulting in deformities and functional disability if not properly managed. Unlike osteoarthritis, which is degenerative in nature, RA is immunologically mediated and systemic, often involving ExRA such as fatigue, anemia, vasculitis, along with cardiovascular complications.[1]

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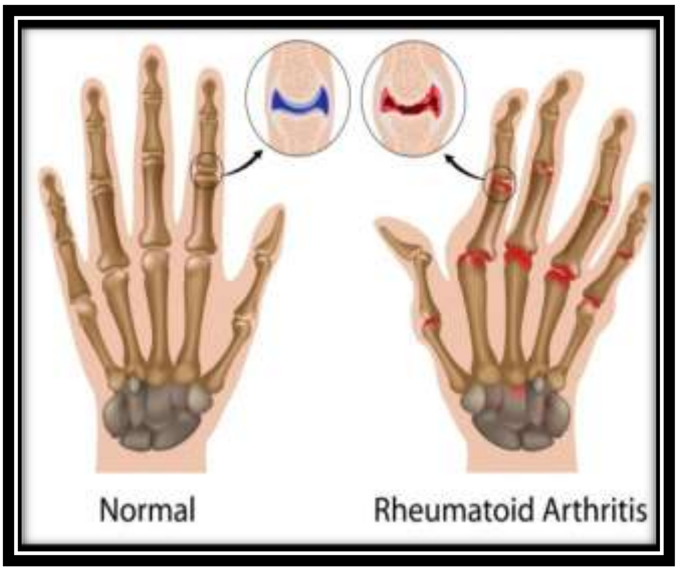

Fig No.1 Internal Structure of Rheumatoid Arthritis

Pathophysiology

Immune dysregulation, environmental triggers, and genetic predisposition interact intricately in the pathophysiology of RA. Rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPAs) are examples of autoantibodies that aid in immune activation. When activated T-lymphocytes, B-cells, and macrophages penetrate the synovium, pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) are overproduced. This mediator stimulatedsynovial hyperplasia, pannus formation, angiogenesis, and activation of osteoclasts, which cause progressive joint destruction.[2]

Symptoms and Clinical Features

Small joints in the hands, wrists, and feet are usually affected by symmetrical polyarthritis when RA patients first arrive. More than an hour of morning stiffness, tenderness, and reduced link function are hallmark features. Extra-articular manifestations may include rheumatoid nodules, pleuritis, pericarditis, neuropathy, and increased risk of cardiovascular disease.[3]

Fig No.2 Symptoms of Rheumatoid Arthritis

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Causes and Risk Factors

The etiology of RA is multifactorial. Genetic susceptibility (e.g., HLA-DR4, HLA-DR1 alleles), hormonal factors, infections, smoking, stress, and environmental triggers contribute to disease onset. The autoimmune response leads to chronic inflammation and progressive joint degeneration.

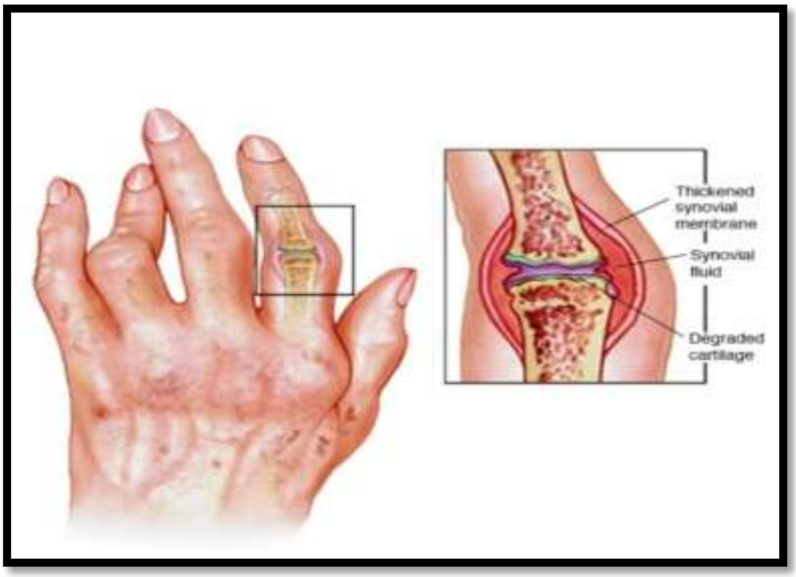

Fig No.3 Cause of Rheumatoid Arthritis

Conventional Therapies and Their Limitation

RA typically involves pharmacological interventions that is cyclooxygenase inhibitors, cortisones, biological agent (biologic DMARDs) like methotrexate, and newer biologics targeting TNF-α and interleukins. While these therapies provide symptomatic relief and slow disease progression, they are often associated with unwanted effects such as stomach disturbance, hepatotoxicity, immunosuppression, osteoporosis, and high treatment costs. Additionally, prolonged use of these substances may make patients less compliant [4].

Role of Herbal Drugs in RA

Due to the drawbacks of conventional therapy, there is increasing interest in complementary and alternative medicine, particularly herbal drugs. Herbal medicines offer multi-targeted mechanisms of action, including anti-inflammatory, antioxidant, analgesic, immunomodulatory, and chondroprotective effects. Several plant-derived compounds such as flavonoids, alkaloids, terpenoids, and saponins act on cytokine pathways, suppress NF-κB activation, and reduce oxidative stress, thereby alleviating joint inflammation and preventing cartilage degradation. Herbs like Zingiber officinale (ginger), Boswellia serrata (shallaki), Curcuma longa (turmeric), and Withaniasomnifera (ashwagandha) have demonstrated significant potential in both preclinical and clinical studies.[5,6]

Pathology of Erosive arthritis:

Erosive arthritis is an immune-mediated disease in which the defence mechanism mistakenly targets the synovial membrane of the skeletal connection, resulting in persistent redness, cartilage damage, also bone deterioration. Its pathogenesis includes a complex correlation of immune mechanisms, surrounding influences, and hereditary predispositions.

1. Initiation (hereditary &Environmental Elements)

Genetic factors: Susceptibility is increased when Human Leukocyte Antigen-DR1and Human Leukocyte Antigen-DR4 alleles are present.

• **Environmental Surrounding:** Stress, hormonal imbalance, bacterial and viral infections, plus smoking all serve as triggers.

• When combined, these cause autoreactive immune cells to become activated and self-tolerance to decline.

2. Immune System Activation

• Thymus derived-lymphocytes, also known as Helper T-cells, enter the synovium and stimulate phagocytes and Bursa ofFabriciusderived-cells.

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• B-units generate autoantibodies like:

- Antibodies against the Fc portion of IgG are known as erosive arthritis factor (RF) antibodies.
- Anti-CCP/ACPAare very specific for chronic polyarthritis.
- Immune-activating cytokines released by antigen presenting cells and phagocytes intensify the immune response. [7]

3. Cytokine Cascade

The activated immune cells release multiple inflammation promoting mediators, like:

• TNF-α, or tumor necrosis factor-alpha
• IL-1, or interleukin-1

Such chemokines activateFLS, endothelial cells and bone resorbing cells,leading for:

- Synovial inflammation
- Angiogenesis (formation of new blood vessels in synovium)
- Recruitment of more inflammatory cells
- Bone resorption[8]

4. Synovial Hyperplasia & Pannus Formation

- Chronic inflammation causes thickening of the synovial membrane.
- This abnormal tissue mass is called pannus, which aggressively invades cartilage and bone.
- Pannus contains macrophages, T-cells, B-cells, fibroblasts, and new blood vessels.
- Release of enzymes (matrix metalloproteinases, collagenases) further damages cartilage.

5. Cartilage Destruction & Bone Erosion

- Activated osteoclasts (stimulated by RANKL from T-cells) cause bone resorption.
- Cartilage breakdown occurs due to continuous pannus invasion and enzymatic degradation.
- Leads to loss of joint space, deformity, and ankylosis (fusion of joints in severe cases).[9]

6. Clinical Outcomes

Joint level: Pain, swelling, morning stiffness, reduced mobility, abnormalities.

Systemic level:

- Fatigue, anemia of chronic disease
- Rheumatoid nodules (subcutaneous)
- Vasculitis, neuropathy
- Pulmonary involvement (pleuritis, interstitial lung disease)
- Cardiovascular complications (↑ risk of atherosclerosis, myocardial infarction)[10]

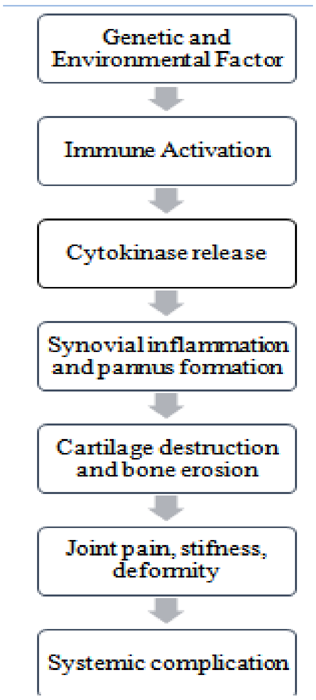

Fig No.4 Pathology Flow Diagram

II. PLANT PROFILE:

1. GINGER:

Fig No.5 Ginger

1.1. Biological Source

Ginger consists of the dried or fresh rhizomes of Zingiber officinale Roscoe.

1.2. Taxonomical Classification

• **Kingdom**	Plantae
• **Division**	Magnoliophyta
• **Class**	Liliopsida
• **Order**	Zingiberales
• **Family**	Zingiberaceae
• **Genus**	Zingiber
• **Species**	Zingiber officinale

Table No. 1. Taxonomical Classification of Ginger

1.3. Common Names: Ginger, Adrak,

1.4. Chemical Constituents

- **Volatile oils (1–3%)**: Zingiberene, β-sesquiphellandrene, borneol, cineole.
- **Non-volatile pungent principles**:
  - ➢ Gingerols (responsible for pungency, anti-inflammatory action)
  - ➢ Shogaols (formed on drying/heating)
  - ➢ Zingerone
- Resins, starch, proteins, fats, minerals.[11]

1.5. Pharmacological Actions / Uses

- **Anti-inflammatory & analgesic** – Inhibits COX and LOX enzymes → useful in RA management.
- **Digestive aid** – Increases gastric motility, relieves nausea, vomiting.
- **Antioxidant** – Scavenges free radicals.
- **Antimicrobial** – Against bacteria and fungi.
- **Cardioprotective** – Improves circulation, reduces cholesterol.
- **In RA** → Reduces pain, stiffness,swelling due to inhibition of prostaglandin and leukotriene synthesis.[12]

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2. ALOE VERA:

Fig No.6 Aloe vera

2.1. Biological Source

- Aloe consists of the dried latex and fresh gel obtained from the leaves of Aloe vera (L.) Burm. f., or other species of Aloe (Fam. Asphodelaceae).

2.2. Taxonomical Classification

• **Kingdom**	Plantae
• **Division**	Tracheophyta
• **Class**	Liliopsida
• **Order**	Asparagales
• **Family**	Asphodelaceae
• **Genus**	Aloe
• **Species**	A. vera

Table No. 2. Taxonomical Classification of Aloe Vera

2.3. Common Names: Aloe vera (English), Ghritkumari (Hindi), Kamari (Sanskrit).

2.4. Chemical Constituents

- **Anthraquinone glycosides**: Aloin A & B (barbaloin) – laxative effect.
- **Chromones**: Aloesin, aloesone.
- **Polysaccharides**: Acemannan (immunomodulatory, wound healing).
- **Sterols**: Lupeol, β-sitosterol, campesterol.
- **Enzymes, vitamins (A, C, E, B12), minerals (Ca, Mg, Zn), amino acids.**

2.5. Pharmacological Actions / Uses

- **Anti-inflammatory** – Inhibits prostaglandins and bradykinin.
- **Wound healing** – Accelerates epithelial regeneration.
- **Laxative** – Due to anthraquinones.
- **Immunomodulatory & antioxidant** – Due to polysaccharides and vitamins.
- **Antimicrobial** – Effective against bacteria and fungi.
- **In RA** → Aloe gel helps reduce joint inflammation, oxidative stress, and provides symptomatic relief.[13]

3. ASHWAGANDHA:

Fig No.7 Ashwagandha

3.1.Common Name: Indian Ginseng, Winter Cherry, Asgandh
3.2.Biological Source: Dried roots and leaves ofIndian Ginseng.
3.3.Taxonomy:

• Kingdom	Plantae
• Division	Magnoliophyta
• Class	Magnoliopsida
• Order	Solanales
• Family	Solanaceae
• Genus	Withania
• Species	Withaniasomnifera

Table No .3. Taxonomical Classification of Ashwagandha

3.4.Chemical Constituents:
- Alkaloids: Somniferine, Withanine, Anaferine
- Steroidal Lactones: Withanolides, Withaferins
- Saponins, Iron, and Flavonoids

3.5.Uses:
- Adaptogen and immunomodulator
- Anti-inflammatory, anti-arthritic, and analgesic
- Enhances physical stamina, reduces stress
- Supports joint health in rheumatoid arthritis[14]

4. GARLIC:

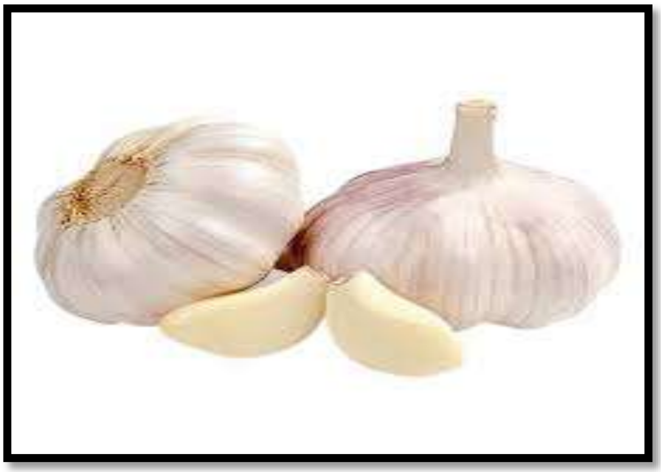

Fig No.8Garlic

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4.1.Common Name: Garlic, Lahsun
4.2.Biological Source: Fresh bulbs of Allium sativum Linn.
4.3.Taxonomy:

• Kingdom	Plantae
• Division	Magnoliophyta
• Class	Liliopsida
• Order	Asparagales
• Family	Amaryllidaceae
• Genus	Allium
• Species	Allium sativum

Table No. 4 Taxonomical Classification of Garlic

4.4.Chemical Constituents:
- Sulfur compounds: Alliin, Allicin, Ajoene, Diallyl disulfide
- Volatile oils, Saponins, Flavonoids, Selenium

4.5.Uses:
- Potent anti-inflammatory and antioxidant
- Lowers cholesterol and blood pressure
- Immunomodulatory and antimicrobial
- Reduces inflammatory mediators in rheumatoid arthritis[15,16]

5. TURMERIC:

Fig No.9 Turmeric

5.1.Common Name: Turmeric, Haldi
5.2.Biological Source: Rhizomes of Curcuma longa Linn.
5.3.Taxonomy:

• Kingdom	Plantae
• Division	Magnoliophyta
• Class	Liliopsida
• Order	Zingiberales
• Family	Zingiberaceae
• Genus	Curcuma
• Species	Curcuma longa

Table No. 5 Taxonomical Classification of Turmeric

5.4.Chemical Constituents:
- Curcuminoids: Bisdemethoxycurcumin, Curcumin, Demethoxycurcumin,
- Volatile oils: Atlantone, Turmerone,Zingiberene
- Proteins, Resins, Starch

5.5.Uses:
- Anti-inflammatory, antioxidant, hepatoprotective
- Inhibits NF-κB, COX, and inflammatory cytokines
- Widely used in rheumatoid arthritis and osteoarthritis
- Promotes wound healing and liver protection[17]

MARKETED HERBAL FORMULATIONS:

Sr.no	**Name of product**	**Image**	**Ingredients**	**Uses**
1.	Rumalaya forte(Himalaya)		Ingredients: Boswellia serrata (Shallaki), Commiphora mukul (Guggulu), Alpinia galanga, Licorice (Glycyrrhiza glabra), Tribulus terrestris.	Anti-inflammatory, relieves pain, improves mobility, prevents joint degeneration.
2.	Baidyanath (Ashwagandha)		Extract of Withaniasomnifera roots.	Immunomodulator, anti-inflammatory, reduces fatigue, improves muscle strength in RA.
3.	Yograj (Guggulu)		Guggulu (Commiphora mukul), Musta (Cyperus rotundus), Chitraka (Plumbago zeylanica), Triphala, Trikatu.	Relieves joint stiffness, inflammation, and pain; detoxifies body; used in chronic arthritis.

4.	Rumalaya (Himalaya)		Boswellia (Shallaki), Spearmint, Myrica esculenta (Gaultheria fragrantissima).	Topical anti-inflammatory gel; reduces pain, swelling, and stiffness in affected joints.
5.	Arthrella		Shallaki (Boswellia), Ashwagandha, Shunthi (Ginger), Nirgundi (Vitex negundo), Guggulu.	Provides pain relief, reduces inflammation, improves joint mobility.
6.	Aethrem		Extract of Artemisia annua.	Reduce inflammation, joint pain, and stiffness in arthritis patients.
7.	Riffway (Joint care)		Shallaki,Rasna, Ashwagandha, Shunthi, Eranda.	Anti-inflammatory, strengthens joints, reduces morning stiffness in RA.

Table No. 6 Marketed Herbal Formulation of Rheumatoid Arthritis

III. CONCLUSION:

Synovial inflammation,connective tissue destruction and systemic complications are hallmarks of symmetric polyarthritiswhich is long standing, continuously increased self-immune disease.

Despite significant advances in conventional treatmentincluding NSAIDs, corticosteroids, DMARDs, and biologic agents, these therapies often come with limitations such as high cost, long-term toxicity, immune suppression, and incomplete remission. Hence, there is a growing interest in exploring herbal drugs as safer, cost-effective, and holistic alternatives or adjuncts in RA management.

A wide range of botanical drug—that are Boswellia serrata (Shallaki), Indian saffron, Giloy, Winter cherry (Ashwagandha),Adrak, and Aloevera—have demonstrated significant prostaglandin inhibitors, redox regulator, as well as immune balancing effects. Their bioactive constituents, including curcumin, boswellic acids, withanolides, and gingerols, act on multiple molecular targets such as TNF-α, IL-1, IL-6, COX, LOX, and NF-κB pathways, thereby reducing inflammation, oxidative stress, and joint damage. Unlike synthetic drugs, herbal remedies often provide multi-targeted effects with fewer side effects and better long-term tolerability.

Moreover, various marketed formulations such as Rumalaya Forte, Yograj Guggulu, SimhanadGuggulu, and Arthrella highlight the integration of traditional knowledge with modern formulations for joint health. However, challenges remain in terms of standardization, quality control, bioavailability enhancement, and large-scale clinical validation of herbal medicines.

In conclusion, herbal drugs offer a promising complementary approach in the management of RA. They not only alleviate indicators that are ache, edema, and rigidity but also target underlying immune-inflammatory mechanisms. Future research should focus on phytochemical standardization, mechanistic studies, advanced drug delivery systems, and well-designed clinical trials to establish evidence-based integration of herbal medicines with conventional therapies. If pursued systematically, herbal drugs may emerge as valuable therapeutic options in providing safer, sustainable, and patient-friendly solutions for rheumatoid arthritis.

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