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A REVIEW ON RHEUMATOID ARTHRITIS

S. Chandra Prakash Reddy*, Sree Mahalakshmi Pasumarthy[1], K. Hena Jyothsna[2], P. Angel[2], M. Pavan Kalyan[2], Sk. Mehaboob Shareef[2], Sreenivasulu Munna[3]

*[1]Assistant Professor, Dept of Pharmacology, Narayana Pharmacy College, Nellore, A.P, India.
[2]Students at Narayana Pharmacy College, Nellore, A.P, India.
[3]Principal, Narayana Pharmacy College, Nellore, A.P, India.

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***Corresponding Author: S. Chandra Prakash Reddy**
Assistant Professor, Dept of Pharmacology, Narayana Pharmacy College, Nellore, A.P, India.
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ABSTRACT

Rheumatoid arthritis is a chronic systemic auto immune disorder characterized by inflammation and progressive damage to joints, particularly the synovial membrane. It affects approximately 1% global population, with a higher incidence in women compared to men. The pathophysiology of rheumatoid arthritis involves the activation of the immune system, leading to the production of pro-inflammatory cytokines such as tumour necrosis factor (TNF), interleukin-6 (IL-6), and interleukin-1, which contribute to joint inflammation and destruction. Early diagnosis and management are crucial to prevent long term disability and improve quality of life. Treatment strategies focus on controlling inflammation through disease- modifying antirheumatic drugs, including biological agents and Janus Kinase Inhibitor (JAK inhibitor). The precise cause of the disease is still unknown despite improvements in treatment options; genetic, environmental, and hormonal variables all have a role. Natural herbs are also used to treat the condition. Research is still being conducted to find new biomarkers for early detection, improve individualized treatment plans, and investigate possible curative treatments. An overview of rheumatoid arthritis's current knowledge, clinical care, and research directions is given in this article.

KEYWORDS: Janus Kinase Inhibitor, Interleukins, Synovial damage, Natural Herbs, Inflammation.

INTRODUCTION

Rheumatoid arthritis is a chronic, systemic, inflammatory autoimmune disease that initially affects small joints, progressing to larger joints, and sometimes the skin, eyes, heart, kidneys, lungs. The bone and the cartilage of joints are damaged, tendon and ligaments become weaken. This damage to the joints cause's deformities and bone erosion, which

is very painful for a patient. The common symptoms of Rheumatoid arthritis also include morning stiffness, fatigue, fever, weight loss. The disease usually starts from the age of 35 to 60 years. It also affects young children less than 16 years of age, referred to as juvenile rheumatoid arthritis which is similar to RA except that rheumatoid factor is not found.[1]

Epidemiology

The main characteristic of rheumatoid arthritis, a systemic illness, is joint inflammation. The distribution of RA in the population and potential risk factors for the onset and progression of the disease have been described using epidemiologic principles. In epidemiological research, the condition may have been defined using a variety of categories, which may have produced different findings. However, researchers may draw contradictory conclusions if the characteristics causing the differences in comparison are not standardized. Mostly researchers use the 1987 American College of Rheumatology (ACR) criteria for the definition of RA and include in studies a sufficient sample size of individuals over 16 years while most recent studies report the prevalence and incidence rates.

Since RA is a flare-and-remit disease, the majority of research on the condition estimates its periodic prevalence. However, each country's unique socioeconomic, demographic, and healthcare circumstances could lead to significant biases. Although rates differ by nation and geographic area, the estimated global prevalence of RA ranges from 0.24 to 1%. In comparison to the prevalence of rheumatoid arthritis, the number of published community-based incidence studies is rather low. Long durations of population follow-up are required for these investigations, and finding sufficient medical data resources is problematic for those who depend on medical records.[2]

Etiology

The exact cause of rheumatoid arthritis (RA) is unknown, but thought to be caused by a combination of factors. These factors include genetics, environmental factors, and sex hormones.

Genetics - Certain genes may increase the risk of developing RA such as TRAF-1, STAT-4, but not everyone with these genes will develop the disease. More than one gene may be involved in determining who gets RA and how severe it will be. A person may be more susceptible to RA if a close relative has it.

Environmental factors: Researchers continue to study how the environmental factors such as cigarette smoking may trigger rheumatoid arthritis in people who have specific genes that also increase their risk. In addition, to some factors such as inhalants, bacteria, viruses, and lung disease may play a role in the development of rheumatoid arthritis. Infections by microorganisms like Porphyromonas gingivalis (P. gingivalis), Proteus mirabilis (P. mirabilis), Epstein–Barr virus (EBV), and mycoplasma may contribute to RA.

Sex hormones: The sex hormones may also play a role in the development of rheumatoid arthritis. Women are more likely affected when compared to men to develop rheumatoid arthritis. According to the Centres for Disease Control and Prevention (CDC), women's have affected more when compared to men. The disease may developed during pregnancy and after pregnancy. High levels of oestrogen a female sex hormone also present in males, may contribute to the development disease.[3]

Testosterone - In the year 2018, researchers published that the results of a study involving 59 participants with rheumatoid arthritis and 61 participants without the condition, matched for sex and age. Those with the rheumatoid

arthritis were more likely to have testosterone levels outside the normal range. Some participants with rheumatoid arthritis then received serum testosterone therapy, and the activity of their rheumatoid arthritis reduced. The authors believe that hormone replacement therapy may help treat symptoms of RA.

Risk Factors

Age - Rheumatoid arthritis can develop at any age, but the risk factors increases as people get older.

Smoking- Smoking may responsible for developing RA, even among people with low-level, lifelong exposure to smoke. Also, the heavy smokers may have more severe rheumatoid arthritis symptoms. Smoking can also cause the oxidative stress and increase the frequency of the body inflammatory response. It can also make some prescription rheumatoid arthritis medications are less effective.

Stress- RA may be influenced by stress. For instance, RA develops as a result of how the body responds to stressful situations. People with the rheumatic conditions often report that their symptoms are appeared shortly after traumatic or stressful experiences, and many people find that stress causes rheumatoid arthritis symptoms to flare up.

Obesity - Obesity with several health issues, such as metabolic syndrome can cause RA symptoms. For example, inflammation is a common feature for both the obesity and metabolic syndrome.

Previous infection - Rheumatoid arthritis may be brought on by an infection's effects on the immune system. The immune system's capacity to combat certain microorganisms, including bacteria or viruses, is compromised. New antigens may be produced as a result of the infection, which also makes the immune system overactive. In a process known as "bystander activation," the immune system's reaction to the virus also targets certain bodily processes.

Gut bacteria- The untreated RA had Prevotella copri bacteria in their intestines. This was present in only 21% of participants in a control group and in only 12% of a group receiving treatment for chronic RA. The researchers proposed that prevotella copri may play a role in inflammation, which can help trigger the rheumatoid arthritis.

Diet- Dietary factors also affect the risk of many diseases, and some researchers have suggested that certain substances in foods can trigger the onset of rheumatoid arthritis.[2]

Symptoms

The main symptoms of the rheumatoid arthritis are joint pain, swelling, stiffness, redness and warmth. It may also cause some general symptoms, and inflammation in other parts of the body. The symptoms of rheumatoid arthritis often develop gradually several weeks. Rheumatoid arthritis mainly affects the joints. These may cause the problems in any joint in the body, although the small joints in the hands and feet are to be affected.

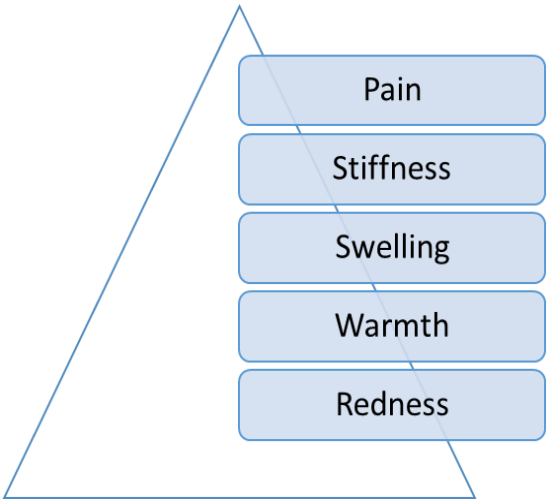

Fig: Symptoms of Rheumatoid Arthritis.

The joint pain may associate with rheumatoid arthritis is usually a throbbing and aching pain. It is often more in the mornings and after a period of inactivity. Joints affected by rheumatoid arthritis can feel stiff. For example, if your hands are affected, difficult to fully bend your fingers or form a fist. The lining of joints are affected by rheumatoid arthritis become inflamed, which can cause the joints to swell. In some people, the swellings called rheumatoid nodules can also develop under the skin around affected joints.

Additional symptoms

As well as the problems affecting the joints, some people with rheumatoid arthritis have more common symptoms are such as:

- Tiredness and a lack of energy
- High temperature
- Sweating
- Poor appetite
- Weight loss[4]

Pathophysiology

Active rheumatoid arthritis is characterized by synovitis, swelling, and joint destruction as the result of a complicated autoimmune and inflammatory process involving both the innate and adaptive immune systems. In a vulnerable patient, the interplay of environment and genes causes a loss of tolerance, leading to rheumatoid arthritis. Rheumatoid arthritis can be initiated by an environmental stimulus in a genetically predisposed host. The finest example is tobacco use in people with the HLA-DRB1 "shared epitome" gene, which results in ACPA-positive rheumatoid arthritis. RF and ACPA antibodies are the most well-known auto antibodies in rheumatoid arthritis. Antibodies against citrullinated proteins are common among rheumatoid arthritis patients. These antibodies have been found in certain rheumatoid arthritis patients since 1964.[4]

A cyclic citrullinated peptide was produced by which can be utilized in an ELISA to test the antibodies in patients in a clinical context. These antibodies are called as anti-cyclic citrullinated peptide antibodies. The peptidyl arginine deiminase post-transcriptionally modifies arginine to produce citrulline. This reaction is supposed to happen where

there is inflammation and tissue damage. The citrulline-containing epitope was preferentially shared by HLA-DRB1. IgG, IgM, or IgA isotypes are ACPA. Citrullinated residues on self-proteins such as type 2 collagen, fibronectin, fibrinogen, and histones can be bound by the ACPA. Rheumatoid arthritis patients also have anti-carbamylated protein antibodies, or anti-CarP antibodies.[5]

In rheumatoid arthritis, the immune response begins at locations that are far from the GI system, lungs, mouth, and synovial joints. The altered proteins in these tissues are created by biochemical processes like citrullination. As an example, smoking cigarettes causes alveolar macrophages to express peptidyl arginine deiminase, which in turn causes arginine to be converted to citrulline in the airway. Anti-citrullinated protein antibodies are produced as a result of this process, which produces a "neoantigen" that stimulates the immune system. The development of an immune response to changed proteins and antibodies against modified proteins is genetically predisposed in patients. Peptidyl arginine deiminase reacts with arginine to produce citrullination. The two Isoforms most closely linked to rheumatoid arthritis are PAD2 and PAD4. Autoimmune antibodies manifest prior to the development of clinical arthritis. Often known as pre-symptomatic or pre-clinical rheumatoid arthritis, the autoimmunity begins at the molecular and cellular level prior to the clinical phase of the disease. Some immunological and biochemical anomalies have been discovered during this stage. Serum levels of RF and ACPA may exist for up to ten years prior to the development of clinical symptoms.[6]

Within time the concentration of ACPA and serum cytokine levels increase. Many patients develop auto antibodies but do not develop the over the disease. Some patients will eventually transition from autoimmunity to immune-mediated inflammation primarily focused in synovium. These auto antibodies are produced by the plasma cells in the synovium. The synovium in rheumatoid arthritis is infiltrated by the immune cells, which include innate immune cells (monocytes and mast cells) and adaptive immune cells (T-helper 1 cells, Th1); T-helper B cells, and plasma cells). Synovial fibroblast-like synovial cells (FSC) are activated. Neutrophils are not present in the synovium but exit from the blood to the synovial fluid.[7]

Granulocyte-monocytes colony-stimulating factor (GM-CSF), interleukin-6 (IL-6), and tumor necrosis factor (TNF) are cytokines and chemokines that stimulate endothelial cells and draw immune cells to the synovial compartment. In the rheumatoid synovium, the FSC transforms into an invasive phenotype. Rheumatoid arthritis's defining characteristic is bone degradation, which is caused by the production of RANKL by inflammatory cells and FSC. Notably, synovial biopsies in arthralgia patients who tested positive for the virus showed no abnormalities. The clinically evident condition is thought to be caused by a second environmental trigger. A damaging inflammatory process starts as soon as this is established. Progressive joint deterioration results from the migration of fibroblast-like synoviocytes (FLS) from one joint to another.[8]

Pharmacological Therapies

Hydroxychloroquinine

Rheumatoid arthritis can be chronically treated with Hydroxychloroquine, an anti malarial medication. This medication may reduce the amount of proinflammatory cytokine-derived secreted by monocytes which is a beneficial effect in treating RA. Common adverse effects include issues with the skin, central nervous system, and gastrointestinal tract. High dosages of this medication are very harmful to the eyes. Patients on this medicine should see an ophthalmologist on a regular basis.[9]

Sulfasalazine

Sulfasalazine is a DMARD commonly used to treat irritable bowel disorder. Rheumatoid arthritis can be treated with this DMARD when used with anti-inflammatory drugs. This medication is used to treat rheumatoid arthritis, although its exact mode of action is unknown. It is believed that sulfapyridine, a decreased version of the drug after delivery, may lower monocyte chemo attractant protein (MCP) and interleukin (IL)-8 secretions. In addition to rash, this medication includes gastrointestinal and central nervous system adverse effects. The medication is generally well tolerated by patients, however because it includes salicylate and sulpha components, it should be avoided by those who are allergic to sulpha.[10]

Non –pharmacological therapy

Exercise Therapy

The relevance of exercise treatment in this patient group is highlighted by the fact that exercise training programs appear to be most beneficial for older, more inflammatory, and less fit RA patients—characteristics that are also present in many D2TRA patients. In addition to rheumatoid arthritis's musculoskeletal symptoms, physical activity is crucial for a number of related conditions that may be more common in the D2TRA group. Exercise programs that strengthen the upper and lower extremities have been shown to improve mental health in addition to muscle strength. Walking-based physical activity has also been shown to improve both the duration and quality of sleep in patients with rheumatoid arthritis, though these studies were carried out in less active RA patients; therefore, more research is required to elucidate this effect in active D2TRA patients.[11]

Psychological interventions

Several psychological interventions have been studied in the treatment of RA. The main techniques are (1) education technique (self-management training, coping skills training, modular behavioural education, patient education), (2) stress management and basic psychotherapies, (3) specific psychotherapies like cognitive-behavioural therapy, emotional disclosure, hypnotherapy.

Dietary intervention

Inflammatory rheumatic disorders, such as RA, have been examined in relation to various diets and nutritional supplements. Although some results were obtained in RA patients with lower disease activity, the Mediterranean diet, particularly when paired with physical exercise, demonstrated some influence on quality of life. Patients with RA benefit from vitamin D supplementation, which has a positive impact on disease activity and co-morbidities, such osteoporosis. There is evidence on the possible advantages of probiotic supplementation in RA, and fish oil supplements and herbal treatments such as Tripterygium wilfordii or seeds containing gamma-linolenic acid have demonstrated some positive clinical effects. Although case series of retrospective and pilot research indicate that weight loss has a positive impact on disease activity, physical functioning, and eating habits, obesity can also be a contributing factor in D2TRA.[12]

Novel targets for Rheumatoid arthritis

An inflammatory condition called rheumatoid arthritis causes synovial joints and bones to become inflamed and eventually break down. Up to 30% of patients still have poorly managed rheumatism despite the wide range of antirheumatic medications. The Janus Kinase (JAK) pathway and the T helper-17 (Th17) system - which includes interleukin (IL)-17, IL-21, IL-22, and granulocyte-macrophage colony-stimulating factor (GM-CSF) - are two

pathways that are thought to be intriguing new therapeutic targets in the field of rheumatology. We also go over the therapeutic potential of small-molecule and biological inhibitors that disrupt these pathways. We will be able to provide more effective and individualized treatment for rheumatoid arthritis patients thanks to developments in combination therapy and biomarker screening.[13]

JAK Inhibitors

Recent recommendations by expert rheumatologists proposed to facitinib as a targeted synthetic DMARD to be used in the treatment of RA after the failure of at least one biological DMARD. To facitinib is the first therapeutic compound that targets the intracellular JAK signalling pathway. JAK is a family of intracellular tyrosine kinases that have a potential role in the transduction of cytokine-mediated signals via the JAK–STAT pathway.

Targeting the Th17 pathway

RA is the IL-17/Th17 pathway. Interleukin (IL-17) is a proinflammatory cytokine that is mainly, but not exclusively, produced by Th17 cells. Following the discovery of the Th17 cell, research over the past decade has focused on both the differentiation process and the effector cytokines of this cell.

Combination treatment and dual inhibitor

There are different combinations of biological to treat are expected to achieve more complete control of joint inflammation and destruction, but the first trials blocking TNF in combination with anakinra have shown that this could cause increased rates of infection. However, TNF and IL-17 seem to be a more promising combination to target in the treatment of rheumatoid arthritis.[14]

Herbs used in treating Rheumatoid Arthritis

Plant Name	Scientific Name	Phyto constituents	Findings	Reference
Myrobalan	Terminalia chebula	Arjun glycoside Cinnamic acid Tri ethyl ester of chebulic acid	By lowering the level of serum tumor necrosis factor, myrobalan's anti-arthritic qualities may help treat rheumatoid arthritis. TNF-alpha and IL-6, two important inflammatory cytokines, are inhibited by Myrobalan, which may also affect immune cell activity.	15
Indian mallow	Abutilon indicum	Stigma sterol Riboflavin Scoparone Thymine Vanillic acid	By inhibiting enzymes, it reveals their mechanism. Abutilon indicum has the ability to suppress the activity of enzymes such as 5-oxygenase, which is essential for the synthesis of leukotrienes and inflammatory prostaglandins.	16,17
Tinospora cardifolia linn	Tinospora gulanchi	Triterpinoids Tinocardifolin Columbine Poly phenols Essential oil Tannins	Inhibits auto immune arthritis by regulating key immune mediators inflammation and bone damage.	18
Ginger	Zingiber officinale	Sesquiterpenes Phenolic acid	Inhibit the production of inflammatory mediators like	19, 20

		Gingerols Bisabolene, Gingerone	prostaglandins and leukotrienes by suppressing enzyme like Cox and 5-lipoxygenase.	
Ashwagandha	Withania somnifera	Alkaloids Withanolides Saponins	Reduces inflammation by modulating the amount of pro-inflammatory mediators.	21
			Administration of Withania somnifera was found to increase the total number of WBC and bone marrow cells, as well as to increase the titre of circulating antibodies and antibody-producing cells and to stimulate the production of immune cells.	
Black pepper	Piper nigrum	Piperen Alkaloid Monoterpines Hydrocarbon such as sabinene, pinene, lanoline, terpene-4-ol.	Piperine inhibits the migration of activator protein 1 but not nuclear factor, into the nucleus in IL 1beta treated synoviocytes.	22, 23
Boswellia serrata	Indian frankincense	Triterpinoids Diterpenoids Poly phenols, Essential oils Tannins	The active compounds in frankincense called boswellic acid, are believed to work by inhibiting the production of inflammatory substance in help alleviate joint pain the body, which could and swelling associated with rheumatoid arthritis.	24
			Boswellia has been used in the treatment of degenerative and inflammatory joint disorders. It reportedly reduces the total white blood cell count in joint fluid and inhibits leukocyte elastase, which is released in rheumatoid arthritis.	25
Deodar cedar	Cedrus deodara	Sesquiterpenes Terpinoids Flavonoids Glycosides	The wood of the deodar tree contain volatile oils that may inhibit the inflammatory response and reduce pain by inhibiting release of histamine.	26,27
Indian bay leaf	Cinnamomm tamala	Geraniol beta-pinene Camphene Beta-caryophellene Limonene p-cynene	Stem of the Cinnamomm tamala contains the bioactive compounds like eugenol and linalool within the leaf, potentially reducing pain and inflammation in the joints by inhibiting inflammatory pathway.	28, 29
Aginbuti	Ammania baccifera linn	Hentriacontine Dotriacontanol, Betulinic acid Lupeol Quercetin Lawsone β-sitosterol Ellagic acid	Inhibition of prostaglandins synthesis modulating of immune response and antioxidant activity.	30, 31
Teak	Tectona grandis	Astectoquinone Betulinic acid Betulinic aldehyde Squalene Acetovanillone Isofuraldehyde	The teak wood contains the bioactive compounds like Astectoquinone and betulinic acid within the wood, potentially	32

		Evofolin Syringaresinol Medioresinol Balaphonin Lariciresinol Zhebeiresinol 1-hydroxypinoresinol	reducing pain and inflammation in the joints and inhibit the inflammatory pathway.	
Shallaki	Boswellia serrata	It contains essential oil, gum and resin. It's essential oil consists of monoterpenes, diterpenes and Sesquiterpenes. Resin portion mainly composed of pentacyclic triterpene acid of which boswellic-acid is the active moiety.	Boswellia serrate also known as salai guggal or Indian frankincense, is used to treat rheumatoid arthritis by reducing inflammation and blocking the production of pro-inflammatory cytokines and also inhibits 5-lipoxygenase this enzyme breaks down polyunsaturated fatty acid into leukotrienes, which are inflammatory molecules that attack joints.	33

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