Journal of Clinical Cases and Reports

ISSN: 2582-0435 Short Communication | Vol 4 Iss S5

Rheumatoid Arthritis - Leading Cause of Bone & Joint Erosion

Disha Sharma* and Sudhanshu Mishra

Department of Pharmacology, MC Saxena College of Pharmacy, India

Correspondence should be addressed to Disha Sharma, msudhanshu22@gmail.com

Received: September 15, 2020; Accepted: September 26, 2020; Published: October 03, 2020

ABSTRACT

Rheumatoid arthritis (RA) is a autoimmune, chronic, inflammatory disorder distressing the joints with severity among patients by affecting the internal organs. RA varies from the symptom duration of fewer than six months in early stage, and more then that is established. The causative agents can be genetics, gender age, and environmental. The initiation of the disease is from the age of 35 years - 60 years with reduction and exacerbation. The pathogenesis of RA is dependent on factors like interleukin-1 (IL-1), interleukin-6 (IL-6), Cytokines tumor necrosis factor-α (TNF-α) IL-17, produced by CD4+ T cells which are formed by macrophages. The initial treatment of RA was based on NSAIDS, DMRDs (Biological and non biological). Another class of drug like corticosteroids, JAK inhibitors which are now also available for adults with severe RA.

KEYWORDS

Rheumatoid arthritis; Inflammatory disorder; Bone erosion; Joint erosion

INTRODUCTION

Rheumatoid arthritis (RA) is an autoimmune disorder that initially affects the coating of the synovial fluid joints and is connected with socioeconomic burdens, progressive disability, and premature death [1]. RA having symptom below six months is distinct as early stage, and when the symptoms revive for more than months, it is known as established form [2]. RA harm the joints but it also affect inner organs, further causing eternal disability in many cases. The causative agents are genetics, gender age, and environmental contact (occupational and air pollutants). Many complexities can lead, like everlasting joint damage requiring rheumatoid vasculitis [3], arthroplasty, and felty syndrome requiring splenectomy if it remains unaddressed [4]. The bone attrition along with several deformities, with pain is caused by the complication in the joints later which results in cartilage and bone destruction of joints, and weakening of tendons and ligaments [3,4]. General symptoms of RA encompass morning rigidity of the exaggerated joints for >30 min, fatigue, fever, mass loss, joints that are gentle, warm and swollen. In later stage rheumatoid nodules area also found under the skin. The initiation of the disorder is generally from the age of 35 years - 60 years with reduction and exacerbation. It at times badly affects the young children even before 16 years of age which is known as juvenile RA (JRA), which is comparable to RA on the rheumatoid factor is not found [5-7].

Citation: Disha Sharma, Rheumatoid Arthritis - Leading Cause of Bone & Joint Erosion. J Clin Cases Rep 4(S5): 8-11.

PATHOGENESIS OF RHEUMATOID ARTHRITIS

Rheumatoid arthritis is characterized by inflammation of the synovial lining due to increase in the number of synoviocytes and number of immune cells. This increment results in hyperplastic, which causes cartilage and bone erosion. In the synovium of RA patients act as macrophages and T cells that create cytokines which supports cell relocation and inflammation. Factors like interleukin-1 (IL-1), interleukin-6 (IL-6), Cytokines tumor necrosis factor-α (TNF-α) which are formed by macrophages. Some other factors like IL-17, produced by CD4+ T cells, are generally involved in causing inflammation and damage of the cartilage [8].

The cytokines activate synoviocytes helps them to proliferate, which escort to the collapse cartilage and synovial tissue, recognized as pannus [9]. Later the immune cells penetrate the joints, declining the synovial hyperplasia. [11] To aggravate the synovial dendritic cells encourage immunity by attracting T lymphocytes and activating antigen-specific T cells and, also, B cells. These auto antibodies break into the joint through recently developed blood vessels and are presently used in the analysis and prediction of RA [10,11] (Figure 1).

ADVANCEMENT OF CONVENTIONAL THERAPEUTICS OF RHEUMATOID ARTHRITIS

Overall division of the RA management techiniques in four stages. First stage of management of musculoskeletal disorders, RA as well as for osteoarthritis. They provide the general symptomatic relief by reducing swelling and pain by inhibiting the cyclooxygenase enzyme which is accountable for inflammation from the disease but they do not have any effect on the agents causing them [12,13]. Second category is corticosteroids which has higher and strong anti-inflammatory effect as well as analgesic effect. As per National Institute for Health and Clinical Excellence (NICE) corticosteroids should be used merely after the relevance of all other management options as it leads to the side effects like severe osteoporosis and cushing's syndrome, fractures, hypertension, weight gain, cataract, etc. Other options are DMARDs (Non-biologic Disease Modifying Anti-rheumatic Drugs (DMARDs) and Biologic Disease Modifying Anti-rheumatic Drugs (DMARDs) for RA treatment. They work against the development of the disease by preventing additional joint obliteration [14]. Another class of drug is JAK inhibitors which are available for adults with severe rheumatoid arthritis [15] (Table 1).

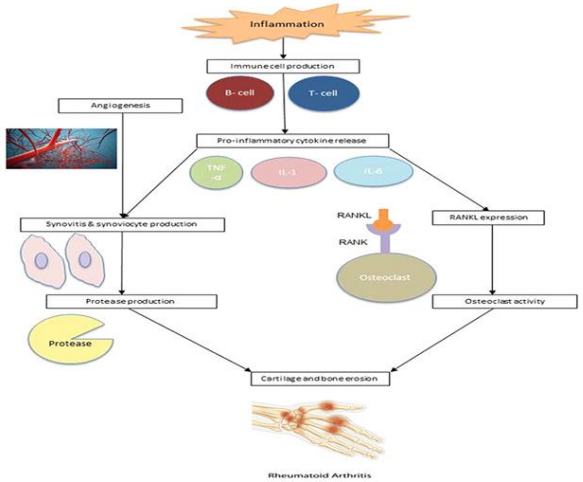

Figure 1: Pathophysiology of RA arthritis.

CONCLUSION

Rheumatoid arthritis (RA) is a persistent, progressive illness that, that eventually leads to joint demolition and disability. The major therapies for rheumatoid arthritis (RA) continue to develop rapidly. There are segregation of novel treatment options, which include biosimilars, JAK inhibitors etc. But it is also important for clinicians to recognize the data concerning drug safety and efficacy. RA treatment areas are accessible; several agents are good only when partially successful or provoke remission in only a marginal number of patients [15]. This scenario has lead has shown high unmet need but in the most recent years, there have been many studies that have enlightened the pathophysiology of RA and offered views on how the disease develops.

Class Name	Drug Name	MOA
NSAIDS	Paracetamol	It is weak inhibitor of PG synthesis of COX-1 and COX-2 in broken cell organization.
	Co-codamol	It is a selective agonist for the mu opioid receptor, but has much weaker affinity to this receptor than drugs like morphine.
	Ibuprofen	It is a non-selective inhibitor of an enzyme called cyclooxygenase (COX), It is necessary for the synthesis of prostaglandins via the arachidonic acid pathway.
Steroids	Prednisolone	It helps to decreases inflammation through suppression of the migration of polymorphonuclear leukocytes and also reversing increased capillary permeability.
Non-biologic DMARD	Methotrexate	It has the pleiotropic therapeutic effects on various immune cells and mediators, consequential in a dampening of the inflammatory response.
	Leflunomide	It helps in reversible inhibition of the mitochondrial enzyme dihydroorotate dehydrogenase (DHODH).
	Sulfasalazine	It is a combination of combination of antibacterial and an anti-inflammatory agent.
Biologic DMARDs: Tumor Necrosis Factor-Alpha Inhibitors (TNFi)	chimeric murine/human IgG1	It is a monoclonal antibody of Infliximab (IFX) that binds to both soluble and membrane-bound TNF-α.
	Etanercept (ETN)	ETN binds to the TNF receptor, preventing TNF-mediated cellular responses.
	Golimumab (GOL)	It is human IgG1 monoclonal antibody neutralizing both soluble and membrane-bound TNF-α.
	Certolizumab Pegol (CZP)	It is a recombinant humanized Fab' fragment of a TNF antibody coupled to polyethylene glycol (PEG).
Biologic DMARDs: Interleukin 1 Inhibitor	Anakinra	It is a recombinant human IL-1 receptor antagonist with a short half-life.
Interleukin 6 and Interleukin 6 Receptor Inhibitors	Tocilizumab (TCZ)	It is the first humanized recombinant IgG1monoclonal antibody that binds to both the soluble and membrane-bound IL-6 receptor, blocking its action and leading to the decrease of the inflammatory response cascade.
JAK inhibitors	Tofacitinib	It is a Janus kinase (JAK3) inhibitor.
	Baricitinib	It is a Janus kinase (JAK1/2) inhibitor.

Table 1: The different category of the drugs used in the management of the RA.

REFERENCES

1. Guo Q, Wang Y, Xu D, et al. (2018) Rheumatoid arthritis: Pathological mechanisms and modern pharmacologic therapies. Bone Research 6(1): 1-14.
2. Chauhan K, Jandu JS, Goyal A, et al. (2020) Rheumatoid arthritis. StatPearls publishing.
3. Lee JE, Kim IJ, Cho MS, et al. (2017) A case of rheumatoid vasculitis involving hepatic artery in early rheumatoid arthritis. Journal of Korean Medical Science 32(7): 1207-1210.
4. Bullock J, Rizvi SA, Saleh AM, et al. (2018) Rheumatoid arthritis: A brief overview of the treatment. Medical Principles and Practice 27(6): 501-507.
5. Fox CQ, Ahmed SS (2002) Physician assistant's clinical review cards. Philadelphia: FA Davis Company: 138-139.
6. McInnes IB, Schett G (2011) The pathogenesis of rheumatoid arthritis. New England Journal of Medicine 365(23): 2205-2219.
7. Chaudhari K, Rizvi S, Syed BA (2016) Rheumatoid arthritis: Current and future trends. Nature Reviews Drug Discovery 15(5): 305-306.
8. Choy E (2012) Understanding the dynamics: Pathways involved in the pathogenesis of rheumatoid arthritis. Rheumatology 51(Suppl_5): v3-v11.
9. Byng-Maddick R, Ehrenstein MR (2015) The impact of biological therapy on regulatory T cells in rheumatoid arthritis. Rheumatology 54(5): 768-775.
10. Boissier MC, Semerano L, Challal S, et al. (2012) Rheumatoid arthritis: From autoimmunity to synovitis and joint destruction. Journal of Autoimmunity 39(3): 222-228.

11. Alwarith J, Kahleova H, Rembert E, et al. (2019) Nutrition interventions in rheumatoid arthritis: The potential use of plant-based diets. A review. Frontiers in Nutrition 6: 141.
12. Schnitzer TJ, Truitt K, Fleischmann R, et al. (1999) The safety profile, tolerability, and effective dose range of rofecoxib in the treatment of rheumatoid arthritis. Clinical Therapeutics 21(10): 1688-1702.
13. Krug H, Broadwell LK, Berry M, et al. (2000) Tolerability and efficacy of nabumetone and naproxen in the treatment of rheumatoid arthritis. Clinical Therapeutics 22(1): 40-52.
14. Kesharwani D, Paliwal R, Satapathy T, et al. (2019) Rheumatiod arthritis: An updated overview of latest therapy and drug delivery. Journal of Pharmacopuncture 22(4): 210-224.
15. (2020) NHS: Treatment; rheumatoid arthritis.