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Rheumatoid Arthritis: A review of current treatment option

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ABSTRACT

Rheumatoid Arthritis (RA) is a chronic autoimmune disease that affects mostly the joints. It causes swelling, pain, stiffness in joints. Recently, disease modifying drugs and the new classes of medications has improved the outcomes that can satisfy most patients. The main aim of the therapy or treatment is achieving the lowest possible levels of arthritis disease activity and remission thereby reducing joint damage, and enhancing physical function and quality of life. The present scenario of treatment requires a comprehensive program which combines medical, social, and emotional support for the patient. There are several classes of drugs used in treatment of RA with some side-effects from which Disease Modifying Anti- Rheumatic Drugs (DMARD's) have shown to alter the disease cause and have an improved radiographic outcomes. Emerging trends in RA treatment, includes mesenchymal stem cell therapies and biosimilar drugs. This comprehensive review aims to provide healthcare professionals and patients with a solid understanding of the current treatment options for RA, making possible of the informed decision-making and good management of the disease.

Keywords: *Rheumatoid Arthritis, medications, therapy, cell therapies, decision-making*

1. INTRODUCTION

According to clinical aspects the presence of signs and symptoms of active joint inflammation is diagnosed as Rheumatoid Arthritis (RA). The symptoms include a swollen joint which is observed by physical examinations and a consistent synovitis. Also biomarkers like autoantibodies and imaging techniques can demonstrate joint inflammation. The diagnosis is termed as "clinical RA" and are categorized into "seropositive" and "seronegative" based on the presence or absence of serum elevations of autoantibodies including Rheumatoid Factor (RF) and/ or anticitrullinated protein antibodies (ACPA).

RA with a symptom duration of less than 6 months is defined as early RA, while if present for more than 6 months, is defined as established RA. While if it is left untreated, it can be a progressive disease with morbidity and increased mortality. The treatment requires both pharmacological and non- pharmacological therapy. At present, the standard of care involves early treatment with DMARD's, but still patients progress to disability and suffer morbidity over time. Therefore, a comprehensive pharmacological and non- pharmacological treatment including counseling, therapy, and patient education is required to improve or enhance the clinical outcomes.

Epidemiology

RA affects 0.5% to 1% of the global population, disproportionately affecting women and increasing in prevalence with age. 'RA' is a " multicasual" disease which is a combination of genetic predisposition and various environmental and lifestyle factors. The Global Burden of Disease 2010 study shows the worldwide prevalence estimate of 0.24%. Epidemiologic estimate of RA and identification of risk factors come largely from the United States or Northern European populations. For health-care planning up-to-date estimates of the burden of RA are required.

Pathophysiology

Genetic Predisposition

RA is a complex autoimmune disease influenced by genetic and environmental factors. Genetic predisposition plays a crucial role, with certain genetic variants increasing the risk of developing RA.

Autoimmune Response

In RA, the immune system mistakenly attacks the lining of the joints (synovium), leading to inflammation and damage. This autoimmune response involves:

1. **Activation of Autoreactive** T Cells: T cells recognize self-antigens, triggering an immune response.

2. **Production of Pro-Inflammatory** Cytokines: Cytokines, such as TNF-alpha, IL-1beta, and IL-6, promote inflammation and joint damage.

3. **Formation of Autoantibodies:** Autoantibodies, like rheumatoid factor (RF) and anti-citrullinated protein antibodies (ACPAs), contribute to the autoimmune response.

Synovial Inflammation

The autoimmune response leads to synovial inflammation, characterized by:

1. **Infiltration of Immune Cells:** Immune cells, such as T cells, B cells, and macrophages, infiltrate the synovium.

2. **Production of Pro-Inflammatory** Mediators: Pro-inflammatory mediators, like cytokines, chemokines, and matrix metalloproteinases (MMPs), promote inflammation and joint damage.

3. **Angiogenesis:** New blood vessel formation contributes to the growth of the synovial pannus.

Joint Damage

Chronic synovial inflammation leads to joint damage, characterized by:

1. **Cartilage Degradation:** Cartilage breakdown occurs due to the production of MMPs and other catabolic enzymes.

2. **Bone Erosion:** Bone erosion results from the activation of osteoclasts and the production of pro-inflammatory cytokines.

3. **Joint Deformity:** Chronic inflammation and joint damage lead to joint deformity and loss of function.

Systemic Involvement

RA is a systemic disease, with potential involvement of:

1. **Extra-Articular Manifestations:** Eye, lung, cardiovascular, and neurological manifestations can occur.

2. **Comorbidities:** RA patients are at increased risk of developing comorbidities, such as cardiovascular disease, osteoporosis, and lymphoma.

Understanding the complex pathophysiology of RA is essential for the development of effective treatment strategies and improving patient outcomes.

Etiology

The etiology of RA is a complex interplay of genetic, environmental, and autoimmune factors. Genetic predisposition plays a crucial role, with specific alleles of the HLA-DRB1 and HLA-DRB4 genes, as well as variants of the PTPN22 gene, increasing the risk of developing RA. Environmental factors, such as smoking, infections, hormonal changes, and dietary factors, can trigger the onset of RA in genetically predisposed individuals. The autoimmune response is characterized by the activation of autoreactive T cells, production of pro-inflammatory cytokines, and formation of autoantibodies, leading to inflammation and damage to the lining of the joints (synovium). Additionally, epigenetic factors, such as DNA methylation and histone modification, and the gut microbiome may also contribute to the development of RA. Understanding the complex etiology of RA is essential for the development of effective prevention and treatment strategies.

Complications

Here are some potential complications of Rheumatoid Arthritis (RA):

Musculoskeletal Complications

1. Joint Deformity: Chronic inflammation can lead to joint deformity and loss of function.

2. Osteoporosis: RA can increase the risk of osteoporosis, particularly in older adults.

3. Tendinitis and Bursitis: Inflammation can affect tendons and bursae, leading to pain and stiffness.

Systemic Complications

1. Cardiovascular Disease: RA increases the risk of cardiovascular disease, including heart attacks, strokes, and peripheral artery disease.

2. Lung Disease: RA can increase the risk of lung disease, including interstitial lung disease and pulmonary nodules.

3. Kidney Disease: RA can increase the risk of kidney disease, particularly in individuals with long-standing disease.

Ocular Complications

1. Dry Eye Syndrome: RA can cause dry eye syndrome, leading to eye discomfort and vision problems.

2. Scleritis: Inflammation can affect the sclera, leading to pain, redness, and vision problems.

3. Uveitis: Inflammation can affect the uvea, leading to pain, redness, and vision problems.

Neurological Complications

1. Carpal Tunnel Syndrome: RA can cause carpal tunnel syndrome, leading to numbness, tingling, and weakness in the hands.

2. Peripheral Neuropathy: RA can cause peripheral neuropathy, leading to numbness, tingling, and weakness in the hands and feet.

3. Atlantoaxial Subluxation: RA can cause atlantoaxial subluxation, leading to neck pain and instability.

Psychological Complications

1. Depression: RA can increase the risk of depression, particularly in individuals with chronic pain and disability.

2. Anxiety: RA can increase the risk of anxiety, particularly in individuals with chronic pain and disability.

3. Fatigue: RA can cause chronic fatigue, leading to decreased productivity and quality of life.

Other Complications

1. Osteonecrosis: RA can increase the risk of osteonecrosis, particularly in individuals with long-standing disease.

2. Infections: RA can increase the risk of infections, particularly in individuals with compromised immune systems.

3. Lymphoma: RA can increase the risk of lymphoma, particularly in individuals with long-standing disease.

Early diagnosis and treatment can help prevent or manage these complications, improving quality of life for individuals with RA.

Treatment:

a. Pharmacological

Here's a comprehensive overview of the pharmacological treatment of Rheumatoid Arthritis (RA):

Disease-Modifying Antirheumatic Drugs (DMARDs)

DMARDs are the cornerstone of RA treatment, aiming to slow disease progression and induce remission. Common DMARDs include:

Methotrexate: First-line treatment for RA, often used in combination with other DMARDs.

Hydroxychloroquine: Used for mild to moderate RA, often in combination with methotrexate.

Sulfasalazine: Used for mild to moderate RA, often in combination with methotrexate.

Leflunomide: Used for moderate to severe RA, often in combination with methotrexate.

Biologic DMARDs (bDMARDs)

bDMARDs are used in combination with conventional DMARDs to achieve better disease control. Common bDMARDs include:

Tumor Necrosis Factor (TNF) Inhibitors: Etanercept, adalimumab, infliximab, certolizumab, and golimumab.

Interleukin-1 (IL-1) Inhibitors: Anakinra.

Interleukin-6 (IL-6) Inhibitors: Tocilizumab.

Janus Kinase (JAK) Inhibitors: Tofacitinib, baricitinib, and upadacitinib.

Targeted Synthetic DMARDs (tsDMARDs)

tsDMARDs are a newer class of medications that target specific molecules involved in the vinflammatory process. Common tsDMARDs include:

JAK Inhibitors: Tofacitinib, baricitinib, and upadacitinib.

Spleen Tyrosine Kinase (SYK) Inhibitors: Fostamatinib.

Glucocorticoids

Glucocorticoids are potent anti-inflammatory medications used to control acute flares and as a bridge therapy until DMARDs take effect. Common glucocorticoids include:

Prednisone: Used for short-term control of inflammation and as a bridge therapy until DMARDs take effect.

Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

NSAIDs are usedc to control pain and inflammation. Common NSAIDs include:

Ibuprofen: Used for pain relief and inflammation control.

Naproxen: Used for pain relief and inflammation control.

Celecoxib: Used for pain relief and inflammation control, with a lower risk of gastrointestinal side effects.

Treatment Strategies

Treatment strategies for RA aim to achieve remission or low disease activity. Common treatment strategies include:

Treat-to-Target (T2T): Aim to achieve remission or low disease activity.

Step-Up Therapy: Start with conventional DMARDs and add biologics or tsDMARDs as needed.

Combination Therapy: Use multiple DMARDs and/or biologics in combination to achieve better disease control.

Monitoring and Adjustment

Regular monitoring and adjustment of treartment are crucial to achieving optimal disease control. Common monitoring tools include:

Disease Activity Score (DAS28): A measure of disease activity based on swollen joint count, tender joint count, erythrocyte sedimentation rate, and patient global assessment.

Clinical Disease Activity Index (CDAI): A measure of disease activity based on swollen joint count, tender joint count, and patient global assessment.

Treatment adjustment is based on disease activity, adverse events and patient preferences.

Non-pharmacological

Here's a detailed overview of non-pharmacological treatments for Rheumatoid Arthritis (RA):

Lifestyle Modifications

- Exercise: Regular exercise, such as yoga, swimming, and cycling, can help improve joint mobility and reduce stiffness.
- Weight Management: Maintaining a healthy weight can reduce pressure on joints and improve overall health.
- Smoking Cessation: Quitting smoking can help reduce disease activity and improve overall health.
- Stress Management: Stress-reducing techniques, such as meditation and deep breathing, can help manage stress and improve overall well-being.

Physical Therapy

- Range of Motion Exercises: Gentle exercises to improve joint mobility and reduce stiffness.
- Strengthening Exercises: Exercises to strengthen muscles around affected joints.
- Physical Modalities: Heat, cold, and electrical stimulation to reduce pain and inflammation.

Occupational Therapy

- Joint Protection Techniques: Techniques to reduce strain on affected joints.
- Assistive Devices: Devices, such as canes and walkers, to reduce strain on affected joints.
- Home Modifications: Modifications to the home environment to reduce strain on affected joints.

Alternative Therapies

- Acupuncture: A technique involving the insertion of fine needles into specific points on the body to reduce pain and inflammation.
- Massage Therapy: A technique involving the manipulation of soft tissue to reduce pain and inflammation.
- Mind-Body Therapies: Techniques, such as meditation and yoga, to reduce stress and improve overall well-being.

Dietary Modifications

- Anti-Inflammatory Diet: A diet rich in fruits, vegetables, and omega-3 fatty acids to reduce inflammation.
- Gluten-Free Diet: A diet that excludes gluten, which may help reduce inflammation in some individuals.
- Vitamin D Supplementation: Supplementation with vitamin D, which is important for bone health.

Assistive Technology

- Adaptive Equipment: Equipment, such as adaptive utensils and assistive devices, to reduce strain on affected joints.
- Mobility Aids: Aids, such as wheelchairs and scooters, to improve mobility and reduce strain on affected joints.

Education and Support

- Patient Education: Education on RA, its treatment, and self-management techniques.
- Support Groups: Groups that provide emotional support and connect individuals with others who have RA.
- These non-pharmacological treatments can help manage RA symptoms, improve quality of life, and reduce the need for medication.

Future Aspects

The future of treatment options for RA holds much promise with the emergence of novel therapies and technologies. Personalized medicine will play a key role, with genetic profiling and biomarker-guided therapy enabling tailored treatment plans. New biologics, such as IL-6 inhibitors and JAK inhibitors, will offer improved treatment results, while small molecule therapies, like kinase inhibitors and phosphodiesterase inhibitors, will provide additional options. Stem cell therapy, gene therapy, and nanotechnology will also become increasingly important, allowing for targeted drug delivery, tissue repair, and regeneration. Further, combination therapies, digital health, and telemedicine will enhance treatment efficacy and patient care. Additionally, advancements in 3D printing, tissue engineering, artificial intelligence, and machine learning will revolutionize the field, enabling personalized treatment plans, predictive analytics, and novel therapeutic approaches.

2. CONCLUSION

Rheumatoid arthritis (RA) is a chronic autoimmune disease with a global presence, often accompanied by various comorbidities and extra-articular manifestations. The incidence and prevalence of RA exhibit significant regional and intra-country variations, attributed to factors such as differences in case definition, population characteristics, socioeconomic and environmental risk factors, and study methodology. RA disproportionately affects women, with the majority of cases manifesting in later adulthood

REFERENCES

[1] Firestein, G. S. (2018). Rheumatoid arthritis: a review of the disease and its treatment. Journal of Autoimmune Diseases, 19(2), 1-13.

[2] McInnes, I. B., et al. (2019). Rheumatoid arthritis: new therapeutic strategies. The Lancet, 393(10171), 484-494.

[3] Smolen, J. S., et al. (2017). EULAR recommendations for the management of rheumatoid arthritis with synthetic and biological disease-modifying antirheumatic drugs: 2016 update. Annals of the Rheumatic Diseases, 76(6), 960-977.

[4] Singh, J. A., et al. (2016). 2016 American College of Rheumatology Guideline for the Treatment of Rheumatoid Arthritis. Arthritis & Rheumatology, 68(1), 1-26.

[5] Smolen, J. S., et al. (2017). EULAR recommendations for the management of rheumatoid arthritis with synthetic and biological disease-modifying antirheumatic drugs: 2016 update. Annals of the Rheumatic Diseases, 76(6), 960-977.

[6] Burmester, G. R., et al. (2019). Targeting cytokines in rheumatoid arthritis: a comprehensive review. Nature Reviews Rheumatology, 15(10), 555-566.

[7] Long H, Liu Q, Yin H, Diao N, Zhang Y, Lin J et al. Prevalence trends of site-specific osteoarthritis from 1990 to 2019: Findings from the global burden of disease study 2019. Arthritis Rheumatol 2022; 74(7): 1172-83.

[8] R. Handa, Management of Rheumatoid Arthritis. Natl Med J India 2004; 17 (3): 143-51

[9] 9. Jennifer N C. Treatment of Rheumatoid Arthritis: A review of Recommendations and Emerging Therapy. Formulary J 2011; 46: 532-45

[10] www. Mayoclinic.org/diseases-conditions/rheumatoid-arthritis/basics/tests-diagnosis/con-20014868(8 Jan 2016)

[11] www.medicinenet.com/rheumatoidarthritis early symptoms /article. htm (9 Jan 2016)

[12] https://en.wikipedia.org/wiki/osteoarthritis#pathophysiology. (9 Jan 2016)

[13] Keith S MD. Osteoarthritis: Diagnosis and Treatment. Am Fam Physician 2012; 85(1): 49-56.

[14] Myasoedova, E.; Davis, J.; Matteson, E.L.; Crowson, C.S. Is the epidemiology of rheumatoid arthritis changing? Results from a population-based incidence study, 1985–2014. Ann. Rheum. Dis. 2020, 79, 440–444. [Google Scholar] [CrossRef]

[15] Roberts-Thomson, P.J.; Jones, M.E.; Walker, J.G.; Macfarlane, J.G.; Smith, M.D.; Ahern, M.J. Stochastic processes in the causation of rheumatic disease. J. Rheumatol. 2002, 29, 2628–2634. [Google Scholar]

[16] Perrot, L.; Hemon, M.; Busnel, J.-M.; Muis-Pistor, O.; Picard, C.; Zandotti, C.; Pham, T.; Roudier, J.; Desplat-Jego, S.; Balandraud, N. First flare of ACPA-positive rheumatoid arthritis after SARS-CoV-2 infection. Lancet Rheumatol. 2021, 3, 6–8. [Google Scholar] [CrossRef]

[17] Elemam, N.M.; Maghazachi, A.A.; Hannawi, S. COVID-19 infection and rheumatoid arthritis: Mutual outburst cytokines and remedies. Curr. Med. Res. Opin. 2021, 37, 1–10. [Google Scholar] [CrossRef]

[18] Favalli, E.G.; Maioli, G.; Biggioggero, M.; Caporali, R. Clinical management of patients with rheumatoid arthritis during the COVID-19 pandemic. Expert Rev. Clin. Immunol. 2021, 17, 561–571. [Google Scholar] [CrossRef]

[19] U.S. National Library of Medicine. A Study of Baricitinib in Participants with Rheumatoid Arthritis (RA-BRANCH). Available online: https://clinicaltrials.gov/ct2/show/NCT04086745 (accessed on 15 October 2021).

[20] U.S. National Library of Medicine. A Study of Baricitinib (LY3009104) in Participants with Rheumatoid Arthritis (RA-BRIDGE). Available online: https://clinicaltrials.gov/ct2/show/NCT03915964 (accessed on 15 October 2021)