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Rheumatoid Arthritis

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ABSTRACTION

A chronic, systemic autoimmune illness that mostly affects joints, rheumatoid arthritis (RA) causes discomfort, inflammation, and eventually joint destruction. The aetiology, pathophysiology, clinical symptoms, diagnosis, and available treatments for RA are all included in this review. Recent developments in personalised medical techniques and biologic medicines are also covered.

1. Introduction

Joint destruction and chronic synovial inflammation are hallmarks of rheumatoid arthritis, an inflammatory illness. About 1% of people worldwide suffer from it, and women are more likely than males to have it. RA increases morbidity and death by affecting not only joints but also extra-articular organs such the heart, lungs, and eyes.

2. Risk factors and aetiology

A complicated interplay between environmental and genetic variables leads to RA. Genetic Factors: RA is closely linked to HLA-DR1 and HLA-DR4. Environmental Triggers: Silica exposure, smoking, and infections (such Epstein-Barr virus) have all been linked. Hormones and Gender: Women are more likely to be impacted, maybe as a result of hormonal factors.

3. Pathophysiology

The development of an aberrant immune response that attacks the synovial membrane is a hallmark of RA. The mechanisms at play are as follows: T-cell and B-cell activation Manufacturing of autoantibodies such as Anti-Citrullinated Protein Antibodies (ACPA) and Rheumatoid Factor (RF) The release of cytokines that promote inflammation, such as TNF-α, IL-1, and IL- Abnormal synovial tissue called pannus forms, invading and destroying bone and cartilage.

4. Diagnosis

Laboratory testing, imaging investigations, and clinical symptoms are used to diagnose RA: Test results for blood: high ESR and CRP, positive RF and ACPA Imaging: MRI, ultrasound, and X-rays reveal synovitis and joint degradation. ACR/EULAR 2010 criteria are commonly utilised.

5. Methods of Therapy

Pharmaceutical Treatment

(a) Corticosteroids and NSAIDs to alleviate symptoms DMARDs, or disease-modifying anti-rheumatic medications, are: Sulfasalazine, Leflunomide, and Methotrexate Pharmaceuticals: JAK inhibitors (Tofacitinib), IL-6 inhibitors (Tocilizumab), B-cell depletion (Rituximab), and TNF inhibitors (Infliximab, Etanercept). Non-pharmacological Physical treatment In occupational therapy Changes in lifestyle (such as stopping smoking and exercising regularly)

b. Surgical Alternatives In extreme situations, joint replacement or synovectomy

6. Current Developments

The treatment of RA has been transformed by biologic treatments. Utilising biomarkers (such ACPA levels) for targeted therapy in personalised medicine Research is being done on gene therapy and stem cell therapy.

7. Complications

If left untreated or not adequately managed, RA can cause: Disability and malformation of the joints Heart-related disorders Osteoporosis. Chronic illness anaemia:

8. Conclusion

A crippling autoimmune condition, rheumatoid arthritis necessitates prompt diagnosis and vigorous therapy. Improvements in biotechnology and immunology have greatly enhanced patient outcomes. It takes a multidisciplinary strategy to control symptoms and avoid long-term issues.

References

1. [04/04/2025, 3:42 p.m.] 1. Smolen, J. S., Aletaha, D., & McInnes, I. B. (2016). +91 70069 26201: 1. An inflammatory condition. 2023–2038; The Lancet, 388(10055). 10.1016/S0140-6736(16)30173-8 has been published.

2. McInnes, I. B., and G. S. Firestein (2017). Rheumatoid arthritis immunopathogenesis. 183–196 in Immunity, 46(2). 10.1016/j.immuni.2017.02.006 at https://doi.org

3. Aletaha, D., Silman, A. J., Neogi, T., et al. (2010). 2010 Classification criteria for rheumatoid arthritis: A collaborative effort between the European League Against Rheumatism and the American College of Rheumatology. 2569–2581 in Arthritis & Rheumatism, 62(9). The article https://doi.org/10.1002/art.27584

4. Rodriguez-Valverde, V., Klareskog, L., van Heijde, der D., et al. (2006). Comparison of methotrexate with etanercept, both separately and in combination, for the management of rheumatoid arthritis. 1287–1295 in The New England Journal of Medicine, 334(2). NEJMoa052730 https://doi.org/10.1056

5. Bridges Jr., S. L., Singh, J. A., Saag, K. G., et al. (2016). 2015 rheumatoid arthritis treatment guidelines from the American College of Rheumatology. 68(1), 1–25; Arthritis Care & Research. 1056/NEJMoa1604028 https://doi.org/10.1002/acr.22783

6. Schett, G., and I. B. McInnes (2011). Rheumatoid arthritis's aetiology. 2205–2219 in The New England Journal of Medicine, 365(23). NEJMra1004965 https://doi.org/10.1056

7. Huizinga, T. W. J., Scott, D. L., and Wolfe, F. (2010). An inflammatory condition. 1094–1108. The Lancet, 376(9746), https://doi.org/10.1016/S0140-6736(10)60826-4

8. Paget, S., Catrina, A. I., and Klareskog, L. (2009). An inflammatory condition. 659–672 in The Lancet, 373(9664), https://doi.org/10.1016/S0140-6736(09)60008-8

9. Yamamoto, K., and R. Yamada (2005). TNF's role in rheumatoid arthritis in animal models. Immunopathology Seminars, Springer, 27(1), 49–62. The article https://doi.org/10.1007/s00281-005-0006-4's

10. Kremer, J., Zamani, O., Genovese, M. C., et al. (2012). Patients with refractory rheumatoid arthritis who use baricitinib. 699–710 in New England Journal of Medicine, 376(7). NEJMoa1604028 https://doi.org/10.1056