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Brief Introduction to Arthritis

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Abstract: *Rheumatoid arthritis is the most common inflammatory arthritis and is a major cause of disability. It existed in early Native American populations several thousand years ago but might not have appeared in Europe until the 17th century. Early theories on the pathogenesis of rheumatoid arthritis focused on autoantibodies and immune complexes. T-cell-mediated antigen-specific responses, T-cell-independent cytokine networks, and aggressive tumour-like behaviour of rheumatoid synovium have also been implicated. More recently, the contribution of autoantibodies has returned to the forefront. Based on the pathogenic mechanisms, specific therapeutic interventions can be designed to suppress synovial inflammation and joint destruction in rheumatoid arthritis(2).*

Objective – Rheumatoid arthritis (RA) is a chronic inflammatory disease affecting diarthrodial joints, inWhich patients tend to perform less physical activity (PA) than recommended. This review focuses onThe existing evidence about the relationship of PA and RA, specifically how the former influences jointInflammation, disability, quality of life and pain in RA patients, and also how disease activity potentiallyImpacts PA in these patients(13).

Ith the growing interest in herbal therapies among persons withRheumatoid arthritis, there exists a need for investigation into their safety andEfficacy. The purpose of this study was to conduct a systematic review to examineThe evidence for the use of herbal medicines for RA based on randomized clinicalTrials (RCTs)(14).

Keywords: *Rheumatoid arthritis*

I. INTRODUCTION

In rheumatoid arthritis the synovial membrane has many of the characteristics of a hyperactive, immunologically-stimulated lymphoid organ. The basis of this hyperactivity is poorly understood. Highly specific antisera to human Ia-like (HLA-DR) antigens and monoclonal antibodies (OKT series) to various T-lymphocyte subsets were used to analyse both the normal and the rheumatoid synovium and to compare it with normal lymph nodes(1) .

Recent years have seen considerable advances in our understanding of both the clinical and basic-research aspects of rheumatoid arthritis. Clinical progress has come from a better recognition of the natural history of the disease, the development and validation of outcome measures for clinical trials and, consequently, innovative trial designs. In parallel, basic research has provided clues to the pathogenic events underlying rheumatoid arthritis, and advances in biotechnology have facilitated the development of new classes of therapeutics. Here, we summarize the fruits of these advances: innovative approaches to the use of existing, traditional disease-modifying antirheumatic drugs; novel agents approved very recently; and further avenues that are presently under investigation or which are of more distant promise(3)

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TABLE 1. Randomized controlled trials for herbal medicines in the treatment of rheumatoid arthritis

First author	Jadad score	Sample size	Intervention/control	Outcome variables
Belch [6]	4	49	540 mg/day GLA in EPO; 450 mg GLA as EPO plus fish oil/liquid paraffin for12 months	Morning stiffness, grip strength, Ritchie AI, pain
Brzeski [7]	4	40	540 mg/day GLA in EPO/ olive oil for 6 months	Ritchie AI, pain, morning stiffness, well-being, health assessment
Chopra [8]	5	182	444 mg RA-1/placebo for 16 weeks	Joint count pain and swelling, health assessment, global assessment
Deal [9]	3	31	0.025% capsaicin cream/ vehicle cream for 4 weeks	Pain, pain relief, global evaluation compared with control group
Deodar [10]	4	18	1200 mg/day curcumin/ 300 mg/day phenylbutazone for 2 weeks	Morning stiffness, walking time, grip strength, articular index, swelling, global assessment
Jäntti [11]	4	18	20 ml/day EPO/olive oil for 12 weeks	Joint score index, pain, morning stiffness, grip strength
Leventhal [12]	4	37	1.4 g/day GLA from borage seed oil/cottonseed oil for 6 months	Joint tenderness count and score, joint swelling count and score, global assessment, pain, morning stiffness, grip strength
Leventhal [13]	5	24	2.0 g/day GLA in BCSO/ placebo tablets containing soyabean oil for 6 months	Joint tenderness count and score, joint swelling count and score, pain, morning stiffness, grip strength, global assessment
Mills [14]	4	20	Reumalex/calcium phosphate for 2 months	Pain, modified Ritchie score
Nordström [15]	3	22	30 g/day flaxseed oil/ safflower oil for 12 weeks	Global assessment, functional class, joint score index pain
Pattrick [16]	4	41	70–86 mm/day feverfew/ cabbage leaf for 6 weeks	Morning stiffness, pain, grip strength, Ritchie AI

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TABLE 1. Continued

First author	Jadad score	Sample size	Intervention/control	Outcome variables
Sander [17]	4	37	1200 mg/day H15 the first week and 3600 or 2400 mg/day for next 11 weeks/placebo tablets for 12 weeks	Ritchie AI for pain and swelling,overall health, pain, NSAID dose
Tao [18]	3	70	60 mg/day TWH/placebo tablets for 12 weeks	Tenderness score, swelling count, morning stiffness, mean grip strength, 15m walking time
Zurier [19]	4	56	2.8 g/day GLA from borage seed oil/sunflower seed oil for 6 months	Swollen joint count, tender joint count and score, pain, degree of disability, duration of morning stiffness, grip strength global assessment

All were double-blind placebo-controlled designs except Deodar [10], which was a double-blind crossover design.

Key points -

Rheumatoid arthritis (RA) is a chronic inflammatory and destructive joint disease that affects 0.5–1% of the population in the industrialized world and commonly leads to significant disability and consequently a reduction in quality of life(3).

Drug therapy for RA rests on two bases: symptomatic treatment with non-steroidal anti-inflammatory drugs (NSAIDs) and disease-modifying antirheumatic drugs (DMARDs). Whereas NSAIDs do not interfere with the underlying immuno-inflammatory events or retard joint destruction, DMARDs, the focus of this review, 'modify' the disease process in all these respects(3).

DMARDs are divided into small-molecule drugs and biological therapies. Approved agents, such as the small molecules methotrexate and leflunomide, and biological therapies, such as tumour-necrosis-factor (TNF) blockers and IL-1 blockers, are briefly reviewed, before considering approaches that could lead to novel agents(3).

Most new candidate small-molecule DMARDs are enzyme inhibitors, which target either secreted enzymes involved in tissue destruction, such as matrix metalloproteinases, enzymes liberating active cytokines from their precursor or membrane-associated forms, or kinases of various signal transduction cascades leading to the activation of transcription factors(3).

Potential approaches to developing novel biological agents that are discussed include targeting TNF, other proinflammatory cytokines and lymphokines; blocking chemokines and angiogenesis; anti-inflammatory cytokines; targeting T cells; targeting B cells and complement; targeting adhesion molecules; targeting Toll-like receptors; and targeting osteoclasts(3).

Side-effects

Lung involvement in RA has several manifestations and is a major cause of morbidity and mortality. The aim of this study was to characterize the different types of lung disease and response to treatment in a UK cohort of RA patients (4).

Rheumatology key messages

Interstitial lung disease was the most common type of lung involvement in RA.

MTX was used in most patients with lung involvement, with acute pneumonitis being extremely rare.

Rituximab demonstrated promising results in RA patients with interstitial lung disease(4).

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Treatment Strategies

In the past decade, there has been a transformation in the Treatment of RA in terms of approach and choice of drugs too.Previous treatment strategies involved initial management By NSAIDs for several years. NSAIDs have represented an Effective therapy for treating RA by eliciting their effects By inhibiting cyclooxygenase activity and blocking the Downstream production of prostanoids and eicosonoids. The Advantages of early management of rheumatoid arthritisWith DMARDs were not well recognized, until recently,And treatment options were limited to mono- or combinationTherapy with a relatively restricted therapeutic armament.DMARD therapy showed decrease in the markers ofInflammation such as erythrocyte sedimentation rate andSwollen joint counts. The combination therapy comprisesImmunosuppressives and DMARDs. This may exacerbateThe potential for hepatic enzyme disturbances. AdditionalSide-effects include weight loss, diarrhea, skin rash andAlopecia [23](15).Although the recent years have brought new informationFor the researchers and clinicians, but the treatment of RAStill remains a challenge. Cytokine research has led to ideaFor the use of anti-cytokine therapy for the treatment of RA.Etanercept (recombinant form of the p75 TNFR-II) andInfliximab (monoclonal antibody directed against TNF-α)Were the first biological response modifiers approved forThe treatment of RA in the year 1992 [24, 25](17,18). Both drugsHave been designed to bind with TNF-α and decrease itsBioavailability. Apart from all the above described treatmentApproaches scientists are now trying to cure RA using geneTherapy, but it is still not fully explored(18).

Alternative Approach

Because of the limitations and risks of conventionalTherapy, people are exploring alternative measures to treatThe disease. Commonly used alternative approaches includeDietary modifications, nutritional supplements and botanicals.The response to these treatments varies from patient toPatient. Alternative treatments have been used both asAdjunct and an alternative to conventional therapy. Most ofThe treatments are relatively free of side effects [26](19).

II. DISCUSSION

Lung disease is one of the extra-articular manifestations of RA that most concerns clinicians, not only due to the wide range of different types of involvement described and the potential increased risk and severity of respiratory infections, but also because of the lack of efficient therapies. Previous studies have already characterized lung involvement in other RA cohorts [4, 19, 20], but in most of them drug-induced lung disease was not described.

Of the different subtypes of lung involvement, RA-ILD was the most prevalent in this cohort, occurring in 51.7% of the patients, which is in line with published data [6, 21, 22]. Despite the reported male predominance in RA-ILD [6, 7, 19, 21], in our cohort 66.7% of the RA-ILD patients were female. This female predominance was even higher in patients with isolated bronchiectasis (87.1%; $P = 0.043$).

Patients with RA-ILD also had a higher frequency of smoking habits and positive RF and ACPA, which is in accordance with the literature [4, 6, 7, 19, 21]. However, three (6.7%) patients with ILD had negative RF and ACPA. Two of these, both ex-smokers, had negative ANA and ENA and presented with NSIP. A third patient was a non-smoker and presented with UIP. This means that despite the postulated role of these autoantibodies in the aetiopathogenesis of RA-associated lung disease [23], patients with negative RF and ACPA are also at risk for lung disease

III. CONCLUSION

Valsartan treatment improved arterial stiffness and CVR in hypertensive patients with RA by normalizing the MCA blood flow velocities in the recovery phase of CO2 test.

ABBREVIATIONS

RA, rheumatoid arthritis; IL-1β, interleukin-1 beta; IL-6,Interleukin-6; TNF-α, tumor necrosis factor alpha; NSAIDs,Non-steroidal anti-inflammatory drugs; DMARDs, diseasModifying anti-rheumatic drugs; ROS, rea

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REFERENCES

[1]. Abrahamsen TG, Froland SS, Natvig JB, Pahle JJ. Antigen and unspecific mitogen stimulation of lymphocytes eluted from rheumatoid inflammatory tissues. Scand j immunol 1976; 5: 1057-63.
[2]. Gary S. Fireste Division of Rheumatology, Allergy and Immunology, School of Medicine, University of California, 9500 Gilman Drive, San Diego, La Jolla, 92093-0656, California, USA
[3]. Josef S. Smole Division of Rheumatology, Department of Internal Medicine III, University of Vienna, and Center of Molecular Medicine, Austrian Academy of Sciences, Vienna, A-1090 Department of Medicine, Center for Rheumatic Diseases, Lainz Hospital, Vienna, A-1130, Austria
[4]. The lung in a cohort of rheumatoid arthritis patients—an overview of different types of involvement and treatment
[5]. Ana C Duarte, Joanna C Porter, Maria J LeandroRheumatology, Volume 58, Issue 11, November 2019, Pages 2031–2038,
[6]. Duarte AC, Sousa S, Cordeiro A, Santos MJ, da Silva JC. FRI0138 Lung involvement in rheumatoid arthritis – a Portuguese reality. Ann Rheum Dis 2017;76:533. Google ScholarWorldCat
[7]. Shaw M, Collins BF, Ho LA, Raghu G. Rheumatoid arthritis-associated lung disease. Eur Respir Rev 2015;24:1–16. Google ScholarCrossrefPubMedWorldCat
[8]. Brown KK. Rheumatoid lung disease. Proc Am Thorac Soc 2007;4:443–8.
[9]. Google ScholarCrossrefPubMedWorldCat
[10]. Zamora-Legoff JA, Krause ML, Crowson CS, Ryu JH, Matteson EL. Patterns of interstitial lung disease and mortality in rheumatoid arthritis. Rheumatology (Oxford) 2017;56:344–50. Google Scholar PubMedWorldCat
[11]. Perez T, Remy-Jardin M, Cortet B. Airways involvement in rheumatoid arthritis. Am J Respir Crit Care Med 1998;157:1658–65. Google ScholarCrossrefPubMedWorldCat
[12]. Kim EJ, Collard HR, King TE. Rheumatoid arthritis-associated interstitial lung disease: the relevance of histopathologic and radiographic pattern. Chest 2009;136:1397–405. Google ScholarCrossref PubMedWorld Cat
[13]. A.Carmona L, González-Alvaro I, Balsa A et al. . Rheumatoid arthritis in Spain: occurrence of extra-articular manifestations and estimates of disease severity. Ann Rheum Dis 2003;62:897–900.
[14]. Google ScholarCrossrefPubMedWorldCat
[15]. a Servicio de Reumatología, Hospital Universitario de Canarias, La Laguna, Spain b Departamento de Medicina Interna, Facultad de Medicina, Universidad de La Laguna, La Laguna, Spain
[16]. K. L. Soeken1,2, S. A. Miller1 and E. Ernst3.
[17]. Moreland, L., Baumgartner, S., Schiff, M., Tindall, E.,
[18]. Fleischmann, R., Weaver, A., Ettlinger, R., Cohen, S.,Koopman, W., Mohler, K., Widmer, M., and Blosch, C.:Treatment of rheumatoid arthritis with a recombinant human Tumor necrosis factor receptor (p75)-Fc fusion protein, N.Eng. J. Med., 337, 141–147, 1997.
[19]. Moreland, L., Michael, H., Scott, W., Elizabeth, A., Roy, M.,Ken, J., Bulpitt, A., Weaver, L., Keystone, E., Furst, D.,Mease, P., Ruderman, E., Horwitz, D., Arkfeld, D., GarrisoL., Burge, D., Blosch, C., Lange, M., McDonnell, N., andWeinblatt, M.: Etanercept therapy in rheumatoid arthritis.Randomised, controlled trial. Ann. Intern. Med., 130, 478– 486, 1999.
[20]. Moreland, L.W., Heck, L.W. Jr., and Koopman, W.J.: Biologic Agents for treating rheumatoid arthritis. Concepts and Progress. Arthritis Rheum., 40, 397–409, 1997.
[21]. Soeken, K., Miller, S., and Ernst, E.: Herbal medicines for The treatment of rheumatoid arthritis: a systematic review Rheumatology, 42, 652–659, 2003.
[22]. Rebrova, Natalia2; Anisimova, Elena1; Ripp, Tatyana1; Mordovin, Victor1; Karpov, Rostislav1; Sarkisova, Olga2; Bogomolova, Irina1