



REVIEW

Hypertension, inflammation and vascular risk in inflammatory rheumatic diseases

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Abstract Vascular risk is higher in patients with rheumatoid arthritis, spondyloarthritis, and psoriatic arthritis than in the general population. In addition to classical vascular risk factors, chronic inflammation plays a crucial role in increasing this risk. In rheumatoid arthritis, the prevalence of hypertension is 41%. Moreover, the prevalence of hypertension has been associated with the duration of spondyloarthritis, as well as the presence of severe psoriasis in psoriatic arthritis. Although some drugs used in the treatment of inflammatory arthritis, such as NSAIDs, glucocorticoids, and Jakinibs, have been associated with an increased vascular risk, their ability to reduce inflammatory activity contributes to a decreased vascular risk in these patients. These drugs should be used with caution, and risk stratification should be performed in patients with vascular risk factors.

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PALABRAS CLAVE

Hipertensión arterial;
Riesgo vascular;
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Espondiloartritis;
AINE;
Glucocorticoides;
Jakinibs

Hipertensión arterial, inflamación y riesgo vascular en las enfermedades reumáticas inflamatorias

Resumen Los pacientes con artritis reumatoide, espondiloartritis y artritis psoriásica tienen un riesgo vascular mayor al de la población general. Además de los factores de riesgo vascular clásicos, la inflamación crónica es un factor crucial en el aumento de este riesgo. En la artritis reumatoide la prevalencia de la hipertensión arterial es del 41%. Asimismo, la prevalencia de hipertensión arterial se ha relacionado con la duración de la espondilitis anquilosante, así como

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la presencia de psoriasis severa en la artritis psoriásica. Si bien algunos fármacos utilizados en el tratamiento de las artritis inflamatorias, como los AINE, los glucocorticoides y los jakinibs se han asociado con un aumento del riesgo vascular, su acción de disminuir la actividad inflamatoria conlleva un descenso del riesgo vascular de este grupo de pacientes. Estos fármacos deben ser indicados con cautela y se debe estratificar el riesgo en pacientes con factores de riesgo vascular.

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Introduction

The vascular risk (VR) of patients with inflammatory rheumatic diseases is higher compared to the general population.¹ This excess VR in rheumatic diseases can be explained due to standard risk factors together with chronic inflammation, which contributes as an independent risk factor and plays a fundamental role in the pathophysiology of cardiovascular disease in rheumatic diseases.^{2–4}

Rheumatoid arthritis (RA) represents the model of inflammatory rheumatic disease. Different studies have shown a higher incidence of cardiovascular events and increased mortality in patients with RA, similar to that which occurs in type 2 diabetes mellitus.^{5,6}

Axial spondyloarthritis (axSpA) is a chronic inflammatory disease that primarily affects the joints of the spine and pelvis. It was known as ankylosing spondylitis when it presented radiographic damage, but today the disease can be identified in its early stages. We refer to radiographic axial spondyloarthritis when there is radiographic damage, or pre-radiographic axial spondyloarthritis when there is no radiographic damage. Both axSpA and psoriatic arthritis (PsA) belong to the group of spondyloarthritis (SpA). These represent a group of chronic inflammatory diseases that share common clinical manifestations, a genetic basis, and pathophysiology. Increased VR has also been observed in these diseases.

The VR of rheumatic diseases has been extensively studied in RA, axSpA, and PsA. The effect of chronic inflammation, combined with standard risk factors and a genetic component, produces endothelial dysfunction, subclinical atherosclerosis, and consequently, cardiovascular events. Furthermore, in PsA, VR is particularly higher in patients with metabolic syndrome as well as in those with significant skin involvement due to psoriasis.⁷

During the last decade, various rheumatology scientific societies have become aware of the need to adequately monitor VR in patients with inflammatory rheumatic diseases and to perform adequate VR stratification.

The objective of this review is to describe the current evidence of increased VR in RA, axSpA, and PsA, with particular emphasis on its relationship with high blood pressure (HBP).

Prevalence of high blood pressure in inflammatory rheumatic diseases

For the optimal management of VR factors in inflammatory rheumatic diseases, and especially hypertension, several studies have been conducted, including the COMORA study,^{8,9} the CARMA study,^{10–12} and the COMOSPA study.^{13–15}

The COMORA study is an international, multicentre, observational study that analysed the prevalence of comorbidities in patients with RA and their management in clinical practice. The results of the Spanish cohort,⁹ which included 200 patients, showed that high blood pressure is one of the most common comorbidities in patients with RA, with a prevalence of 41% of cases. This prevalence was similar in the international cohort, which included 17 countries.⁸

The CARMA (CARdiovascular in RheuMATology) project is a 10-year prospective multicentre study designed to evaluate VR in patients with RA, axSpA, and PsA compared with healthy control patients from rheumatology outpatient clinics without inflammatory disease.

The baseline data from this study, published in 2015, showed that the prevalence of hypertension was 30.5% in patients with RA, 25.7% in axSpA, and 29.5% in PsA.¹⁰ This prevalence increased substantially when analysed in patients who had experienced a cardiovascular event: 65.1%, 63.2%, and 80%, respectively. Subsequently, at 2.5 years of follow-up, the incidence of a first cardiovascular event was analysed.¹¹ The overall result was 15.3 cases per 1000 patients and was observed to be higher in patients with axSpA, men, older patients, patients with hypertension, and patients with long-standing rheumatic disease. The prevalence of hypertension in patients with a first cardiovascular event was 41.5% compared with 28.8% in patients without a cardiovascular event.

At 5 years of follow-up in this CARMA cohort, the incidence of a first cardiovascular event and mortality from this cause were analysed.¹² Thus, it was observed that 86 patients experienced a first cardiovascular event and 11 of 2171 patients analysed died from a cardiovascular event. The group with the highest incidence of a first cardiovascular event was PsA, followed by RA and finally axSpA. However, when calculating this incidence adjusted for age, sex and vascular risk factors, axSpA was the inflammatory rheumatic disease with the highest incidence. Furthermore, the factors that increased the risk of a first cardiovascular event

were patients with axSpA, older patients, patients with hypertension and longer duration of inflammatory rheumatic disease. The cardiovascular mortality observed at 5 years of follow-up was lower than expected. It is believed that the low number of fatal cardiovascular events could be due to the probable protective effect of biological treatment (>40% of patients were on biological treatment). Another factor that could have contributed to the reduction in cardiovascular risk could be the impact of the EULAR (European League Against Rheumatism) recommendations on the management of VR in patients with inflammatory rheumatic disease implemented in clinical settings (discussed below).

As mentioned above, the CARMA study is a 10-year prospective study, and it will be vital to understand the evolution and prognosis associated with VR in these patients.

Similar to the COMORA study, the COMOSPA study is an international observational cross-sectional study that aimed to analyse the prevalence of comorbidities in patients with SpA, including axSpA, PsA, reactive arthritis, and arthritis associated with inflammatory bowel disease.¹³ Initial data showed that hypertension was the most prevalent VR factor (33.5%). Recently, an association between hypertension and the duration of SpA has been published, especially in cases with axial involvement as well as in the subgroup of patients with delayed diagnosis. Previous publications have already suggested that hypertension is more prevalent in axSpA. This association, paradoxically, has not been attributed to the use of NSAIDs.¹⁴

Finally, there is data on the influence of phenotypes (axial or peripheral) and the presence of psoriasis on the prevalence of comorbidities associated with SpA. The peripheral phenotype with psoriasis was independently associated with an increased prevalence of VR factors and was shown to be the group with the highest prevalence of hypertension (44.9%). The presence of psoriasis, per se, is associated with a higher prevalence of VR factors as well as an increased coexistence of metabolic syndrome.¹⁵

Inflammation and autoimmunity

The treatment of inflammatory rheumatic diseases has made significant progress in recent years. However, these diseases are associated with an increase in VR, and therefore with a higher incidence of events and increased cardiovascular mortality. This is partly due to a higher prevalence of hypertension, dyslipidemia, and other VR factors, which are underdiagnosed and undertreated. The higher prevalence of classic VR factors does not fully explain this increased vascular risk, since it is partly due to inflammatory and immunological mechanisms, which accelerate the vascular atherosclerotic process in these patients.^{2,3}

The acute inflammatory reaction is a protective process, which is mostly self-limited. In humans, various processes can occur that provoke sustained inflammatory stimuli, leading to a reduction in counter-regulatory mechanisms, which hinders the return to normal homeostasis.^{3,16} Table 1 shows the different factors that may be associated with an alteration in the resolution of the acute inflammatory phenomenon.

Monocytes and macrophages are crucial elements involved in chronic inflammation, including atherosclerosis

Table 1 Factors associated with failure to resolve the acute inflammatory process.

- Smoking
- Stress
- Sleep disturbances
- Advanced age
- Unhealthy diet
- Sedentary lifestyle
- Pollution

and tissue damage associated with autoimmunity. Published studies have shown that the number of monocytes, especially CD14++CD16, correlates with the onset of cardiovascular events,¹⁷ and an increase in this monocyte subtype has also been observed in RA and other autoimmune diseases.¹⁶ When a cardiovascular event occurs, such as acute myocardial infarction (AMI), the initial acute inflammatory process often fails to resolve, which would lead to an acceleration of the atherosclerosis process.¹⁸ AMI accelerates pre-existing atherosclerosis in non-infarcted vessels by modulating innate and adaptive immune responses, and this is associated with an increased risk of recurrence.¹⁹

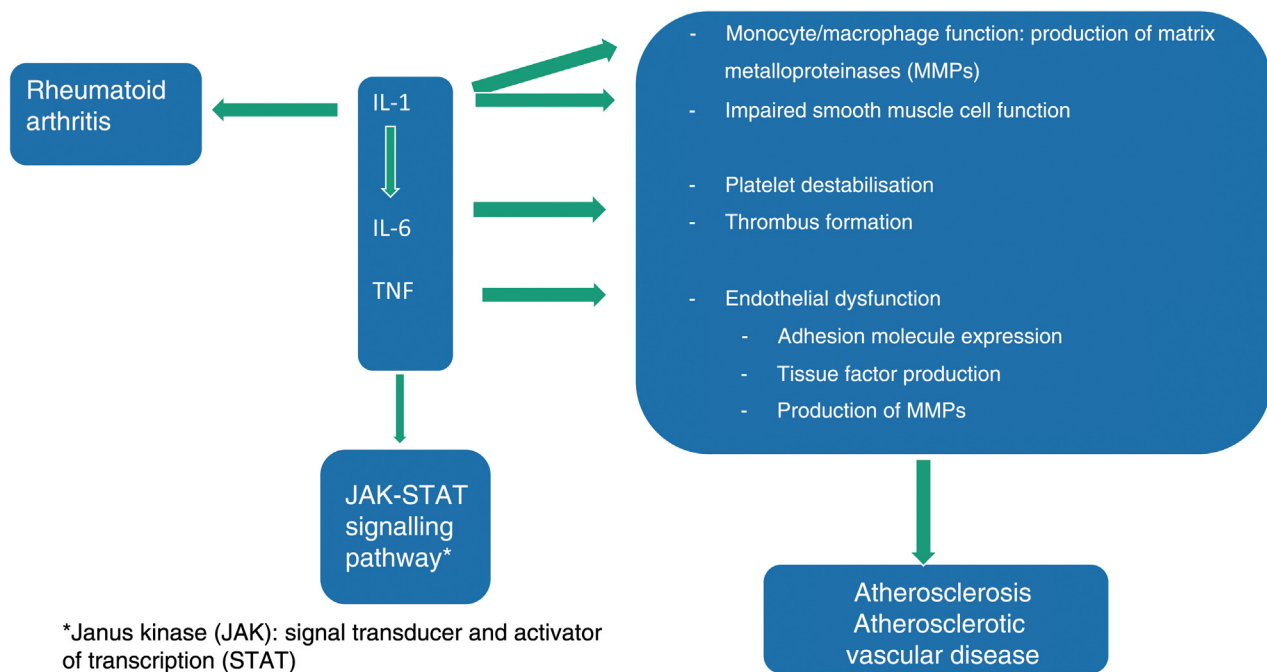
It is well known that RA (the most studied in this context) presents a 50% increase in VR compared to the general population.²⁰ This increased risk is similar to that of patients with type 2 diabetes mellitus.² Increased VR has also been observed in other inflammatory rheumatic diseases.

Similar inflammatory mechanisms coexist in the pathogenesis of RA and atherosclerosis.²¹ Among these, the role of interleukin-1 (IL-1), interleukin-6 (IL-6), and the TNF and JAK-STAT pathways are noteworthy (Fig. 1). Based on current knowledge, the cytokine that plays the most significant role is IL-6.^{21,22} Recent data from the Atherosclerosis Risk in Communities (ARIC) study have shown an association between interleukin-6 (IL-6) and interleukin-18 levels and cardiovascular morbidity and mortality.²³ Furthermore, the Multi-Ethnic Study of Atherosclerosis (MESA) study observed an association between elevated IL-6 levels and the development of cardiovascular events, even in patients with low high-sensitivity CRP levels.²⁴

Inflammatory rheumatic diseases and other autoimmune diseases are frequently accompanied by HBP, which can have direct effects on endothelial cells and vascular reactivity, associated with greater target organ involvement.^{25,26} Furthermore, chronic HBP disrupts the integrity of the blood-brain barrier and the cerebral microvasculature, with an increased risk of brain damage and cognitive decline. Inflammatory phenomena and activation of the autoimmune system also play a role in this process.²⁷

Several studies have shown that these inflammatory mechanisms can begin early, potentially predicting the future development of hypertension. In a meta-analysis Caiazzo et al.²⁸ showed that the level of circulating cytokines is associated with a higher risk of developing HBP.

A better understanding of these inflammatory and immunological mechanisms would help us better understand the effects of the different drugs used to control RA and other inflammatory rheumatic diseases, as well as help us search for new therapeutic targets specifically targeting inflammatory and immunological phenomena, which



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Figure 1 Common inflammatory mechanisms involved in the pathogenesis of rheumatoid arthritis and atherosclerosis.

could improve VR by reducing the process that accelerates atherosclerosis in these patients.^{29,30}

Detection of vascular lesion

There are no specific guidelines for the use of imaging tests to assess VR in patients with inflammatory rheumatic diseases. Considering that VR is increased in this group of patients and that the available algorithms for measuring this risk tend to underestimate it, screening to rule out the presence of asymptomatic atherosclerotic plaques using carotid Doppler ultrasound is recommended. The significance of the presence of altered intima-media thickness for reclassifying VR is a hotly debated topic. However, the presence of carotid atheromatous plaques indicates a very high VR.^{31,32}

Another imaging technique, coronary artery CT, provides precise information on the quantification of coronary calcium and the severity of coronary atherosclerosis. Although not routinely used in clinical practice, it can help stratify VR in some patients.³³

Influence of rheumatic disease treatment on vascular risk

The treatment of RA is based on the "Treat-Target" (T2T) strategy. This strategy consists of achieving a therapeutic objective, such as the remission of inflammatory activity in early arthritis or, in its absence, achieving low inflammatory activity in arthritis. On the other hand, the treatment of SpA is based on achieving remission, or failing that, low activity, of the different affected domains. These domains are peripheral arthritis, axial involvement,

enthesitis, dactylitis, cutaneous domains (psoriasis and onychopathy), inflammatory bowel disease, and uveitis.

It is important to note that in the therapeutic management of RA, axSpA and axSpA share signalling pathways, different therapeutic targets, and mechanisms of action.

Currently, the drugs available globally for the three diseases are: non-steroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, conventional synthetic disease-modifying antirheumatic drugs (DMARDs) (methotrexate, leflunomide, hydroxychloroquine, sulfasalazine), biological DMARDs (anti-TNF α : adalimumab, etanercept, golimumab, certolizumab and infliximab, anti-IL 6: tocilizumab, and sarilumab, anti-CD20: rituximab, anti-CTL4: abatacept, anti-IL 17: sekukinumab, ixekizumab, anti-IL17A/17F: bimekizumab, anti-IL 23: guselkumab and risankizumab, anti-IL12/23: ustekinumab) and synthetic targeted DMARDs, JAK inhibitors or Jakinibs (tofacitinib, baricitinib, upadacitinib, filgotinib).

The potential impact of different drugs on cardiovascular risk is summarised in Table 2.

Among the different groups of drugs used in the treatment of rheumatic diseases, NSAIDs, glucocorticoids, and Jakinibs are particularly relevant in VR, and will therefore be discussed in this review.

The use of NSAIDs and glucocorticoids is essential in the treatment of inflammatory rheumatic diseases for optimal control of inflammatory activity, especially in the early stages. However, it is well known that both types of drugs are associated with an increase in VR and should be used with caution in patients with a high baseline VR. Their prolonged use can produce multiple adverse effects such as hypertension, insulin resistance, dyslipidemia, and mineralocorticoid effects. Furthermore, the effect of these drugs

Table 2 Effect of treatments for inflammatory rheumatic diseases on vascular risk.

Type of drug	Indication in inflammatory rheumatic disease	Increases vascular risk
<i>NSAIDs</i>	RA, r-axSpA, PsAs	+
<i>Glucocorticoids</i>	RA, SpA (peripheral forms), PsAs	+
<i>Conventional synthetic DMARDs</i>		
Methotrexate	RA, SpA (peripheral forms), PsAs	—
Leflunomide	RA, PsAs	+
		(increased blood pressure)
Hydroxychloroquine	RA	—
		(benefits lipid profile)
Sulfasalazine	RA, r-axSpA, PsAs	—
<i>Biological DMARD</i>		
Anti-TNF α	RA, R-axSpA and nr, PsAs	—
		(contraindicated in heart failure)
Anti-IL-6 (tocilizumab, sarilumab)	RA	—
Anti-CD20 (rituximab)	RA	—
Anti-CTL4 (abatacept)	RA	—
Anti-IL17 (ixekizumab)	R-axSpA and nr, PsAs	—
Anti-IL 17A/17F (bimekizumab)	R-axSpA, PsAs	—
Anti-IL23 (guselkumab, risankizumab)	PsAs	—
Anti-IL12/23 (ustekinumab)	PsAs	—
<i>Targeted synthetic DMARD</i>		
Jakinibs		+
Tofacitinib	RA, R-axSpA, v	
Upadacitinib	RA, R-axSpA, PsAs	
Baricitinib	RA	
Filgotinib	RA	

nr-axSpA: non-radiographic axial spondylarthritis; PsA: psoriatic arthritis; RA: rheumatoid arthritis, r-axSpA: radiographic axial spondylarthritis.

+: increases vascular risk; —: no effect on vascular risk.

in reducing the inflammatory activity of rheumatic disease simultaneously contributes to reducing VR.

NSAIDs can be classified into two groups: traditional NSAIDs that inhibit COX-1 and COX-2, and COXIBs that are selective COX-2 inhibitors. Multiple studies have linked NSAIDs to an increased risk of cardiovascular disease through various mechanisms, including fluid retention, increased blood pressure, and increased atherosclerosis or destabilisation of existing plaques.^{34,35}

Due to their mechanism of action, COXIBs are associated with a higher VR compared to traditional NSAIDs. Among these, celecoxib could be considered the safest in this group given its shorter half-life compared to other COXIBs.

The European Medicines Agency (EMA) has confirmed that naproxen and diclofenac are the NSAIDs with the lowest and highest vascular risk, respectively.

An interesting finding is that NSAID use in RA patients may have less impact on VR compared to the general population due to their effect on controlling chronic RA inflammation.³⁶

Recently, EULAR published a systematic review of the literature to assess the efficacy, duration of use, and safety of glucocorticoids.³⁷ In assessing safety, and specifically hypertension and cardiovascular disease, they included 11

observational studies that showed conflicting data. Four studies reported an increased risk of hypertension; in one of these, this increase was associated only with prednisone doses ≥ 7.5 mg/day and for cumulative doses between 5000 and 10,000 mg. In other studies, the increase was independent of the daily dose and cumulative dose. Similarly, studies evaluating increased VR showed conflicting data depending on the glucocorticoid dose.

Given this scenario, EULAR recommends limiting the use of glucocorticoids to the lowest effective dose (7.5 mg/d or lower) and for the shortest possible duration and their rapid withdrawal when possible.

The treatment of RA, axSpA, and PsA has improved significantly since the advent of biologic therapies more than two decades ago, and even more so following the recent introduction of Januskinase inhibitors (Jakinibs). Their introduction has led to improved remission rates, radiographic progression, and patient quality of life.

The initial pivotal studies conducted for the approval of the first marketed Jakinib, tofacitinib, did not identify differences in the risk of MACE (major adverse cardiovascular event) compared to anti-TNF α drugs.³⁸ However, the study evaluating the cardiovascular safety of baricitinib found,

in a sample of selected patients with no prior history of cardiovascular events, that RA patients treated with baricitinib 4 mg/d had a higher risk of venous thromboembolic events.³⁹ This risk was higher in patients with a history of previous venous thromboembolic events, cardiovascular events, overweight, hypercoagulable states, or cancer. A post-marketing study, ORAL Surveillance, was subsequently conducted to further examine safety data, including cardiovascular risk and venous thromboembolic events. Patients with RA, those over 50 years of age, and one or more VR factors were included. Post-hoc analyses of this study showed an increased risk of MACE and venous thromboembolic events, particularly in patients over 65 years of age, with a history of cardiovascular disease, smokers, with active RA, and receiving suboptimal treatment for cardiovascular comorbidities. These results prompted the EMA to issue a statement in 2021 on risk stratification and the cautious use of tofacitinib. In patients over 65 years of age, smokers or former smokers who have smoked for a long time, with a history of cardiovascular events or other cardiovascular risk factors, the use of tofacitinib is only possible if no other therapeutic alternative is available. After one year, the EMA extended these recommendations as a class effect to the other Jakinibs.

Jakinibs, due to their mechanism of action, increase HDL cholesterol (HDL-C) and LDL cholesterol (LDL-C) levels as they decrease systemic inflammation and disease activity. This increase has not been associated with increased VR and can be effectively treated with statins.

This scenario was previously studied more than 20 years ago with the emergence of anti-TNF α agents and their paradoxical effect on cholesterol levels and its fractions. The reduction in the potential inflammatory burden thanks to anti-TNF α agents was more effective than the described effect itself and confirmed their safety and efficacy. Currently, having overcome initial doubts, anti-TNF α agents are the first-line biologic drugs in the treatment of multiple chronic inflammatory processes.

Clinical management of vascular risk in rheumatic diseases

More than a decade ago, EULAR published a specific document with recommendations for the clinical management of VR in patients with RA and other forms of inflammatory arthritis, including axSpA and PsA. An update of this document was published in 2017.⁴⁰ Members of various disciplines, including rheumatologists, cardiologists, internists, epidemiologists, and patient representatives, participated in the development of this document, and it was very useful in the overall management of patients with inflammatory arthritis.

The document is based on the recommendations summarised below. The first necessity is optimum control of the inflammatory activity to reduce VR in these patients. The evolution of therapy for these diseases with the emergence of biologic DMARDs and targeted synthetic DMARDs, as well as the "Treat-to-Target" (T2T) strategy, has substantially improved the control of inflammatory activity and, therefore, cardiovascular risk. It is recommended that VR assessment be performed at least once every 5 years or when

a major change in underlying treatment is made, such as the initiation of biological DMARDs. In this comprehensive management and monitoring of the CV effects of these therapies, close collaboration with advanced practice nursing and the application of SCORE indices validated in populations with inflammatory diseases are of vital importance.⁴¹

Furthermore, EULAR recommends that VR stratification should be based on national guidelines. The latest European guidelines for vascular prevention published in 2021⁴² recommend that VR estimation be performed according to the SCORE2 in patients under 70 years of age, and by the SCORE2 OP in subjects ≥ 70 years of age. The Spanish Interdisciplinary Committee for Vascular Prevention (CEIPV), a body comprised of 14 scientific societies and supported by the Carlos III Health Institute and the Ministry of Health, has adapted these guidelines for national implementation, based on SCORE2 and SCORE2 OP for low-risk countries.^{42,43} The recommendations of these guidelines are that in subjects with RA, the VR estimate obtained using SCORE2/SCORE2 OP should be multiplied by 1.5. This is likely applicable to other inflammatory rheumatic diseases, but the best available evidence is for RA. More recently, EULAR published a new document, but this was more focused on VR management in SLE and other autoimmune diseases.⁴⁴

In inflammatory rheumatic diseases, lipid profile determination is necessary when inflammatory activity is stable or in remission. The phenomenon of paradoxical lipid profile behaviour that can occur in RA after the initiation of biological treatment enables us to observe that patients with high inflammatory activity may present low total cholesterol and LDL-C levels, while their VR actually remains elevated and normalises after the effect of the initiated drug.

EULAR recommends screening for plaque detection using carotid Doppler ultrasound in VR assessment. The presence of plaques reclassifies patients with intermediate VR to very high risk, with the associated therapeutic implications.

As general measures, healthy lifestyles such as regular exercise, a healthy diet, smoking cessation, etc., should be recommended.⁴³ Regarding antihypertensive and lipid-lowering treatment and management of other risk factors, it is recommended to follow the indications of the national and international guidelines contained in a recent document.⁴⁵

In general, the therapeutic goal in patients with hypertension is a systolic BP <130 and a diastolic BP between 70 and 80 mm Hg, especially in those <65 years of age. The same therapeutic goal is also recommended for patients aged 65–79 years if well tolerated. In patients over 80 years of age or in frail patients, a SBP <150 mm Hg may be acceptable, or less than 140 mm Hg if tolerated. A SBP <120 mm Hg or a diastolic BP <70 mm Hg is not recommended. In elderly patients with isolated systolic hypertension, it is recommended to initiate or intensify antihypertensive treatment when SBP is ≥ 160 mm Hg, even if diastolic BP is <70 mm Hg.

Regarding lipid-lowering therapy, the therapeutic goal will depend on the estimated VR.^{42,43} In high-risk patients, the therapeutic goal is to achieve an LDL-C <70 mg/dL and at least a 50% reduction from baseline LDL-C values. In very high-risk patients, the goal is to achieve an LDL-C <55 mg/dL and also a reduction of at least 50% from baseline values. Initial treatment is statins (high intensity in

high or very high-risk cases), and if the therapeutic goal is not achieved, ezetimibe is added. In very high-risk or secondary prevention situations, the therapeutic goal is not always achieved with these drugs, and in these cases, it is recommended to refer the patient to a specialised unit for evaluation of other drugs, such as PCSK-9 inhibitors or bempedoic acid. In patients with hypertriglyceridaemia, the therapeutic approach is to reduce VR by controlling LDL-C (in this case, non-HDL-C). In subjects with severe hypertriglyceridaemia (>1000 mg/dL), reducing triglycerides through lifestyle changes (diet and reducing or completely eliminating alcohol intake) is a priority. Specific drug indications can be found in the aforementioned document.⁴⁵

Finally, when prescribing NSAIDs, they should be used with caution. It is recommended that their use be individualised, taking into account the patient's cardiovascular risk. The use of naproxen is suggested as the NSAID with the safest cardiovascular profile, and failing that, the use of COX-2 inhibitors as more selective and less gastrointestinal-damaging drugs. Likewise, the use of glucocorticoids should be limited to the lowest effective dose and for the shortest possible duration. It is important to bear in mind that good control of inflammatory activity with these drugs can, in turn, have a beneficial effect by reducing the VR.

To sum up, these guidelines emphasize the need for screening, evaluation, and management of VR factors, as well as patient education regarding treatment adherence and lifestyle changes.

Conclusions

Patients with inflammatory rheumatic diseases have a higher VR than the general population. In addition to traditional VR factors, chronic inflammation is a key factor in increasing this risk. RA has been the most studied inflammatory rheumatic disease in this regard. The prevalence of hypertension in RA has been estimated at around 41%. Furthermore, it has been observed that the disease duration of patients with axSpA, as well as the presence of severe psoriasis in patients with PsA, contribute to the increased prevalence of hypertension and other VR factors.

NSAIDs and glucocorticoids have been associated with an increase in VR and should be used with caution in patients with high VR. However, good control of the inflammatory activity of the disease results in a decrease in VR. The emergence of treatments such as biologic DMARDs more than 20 years ago or Jakinibs, more recently, has represented a paradigm shift in the treatment of inflammatory rheumatic diseases. Jakinibs have been linked to an increased incidence of MACE and venous thromboembolic events in a high-risk group of patients. Despite their proven efficacy, their use should be avoided in this subgroup of patients.

It is essential to estimate and stratify VR in these patients periodically, at least every 5 years. It is recommended that strategies to reduce VR in rheumatic diseases become routine for better patient management.

Conflict of interest

The authors declare that they have no conflict of interest.

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