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ANALYSIS OF CARDIOVASCULAR RISK ASSOCIATED WITH JANUS KINASE INHIBITORS TREATMENT IN PATIENTS WITH RHEUMATOID ARTHRITIS

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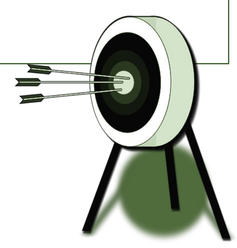
Background and importance

Rheumatoid arthritis (RA) is associated with an increased risk of cardiovascular morbidity and mortality, possibly due to chronic systemic immune-mediated inflammation. Despite increasing evidence of increased risk of cardiovascular events from Janus kinase (JAK) inhibitors, the association between them is unclear.

Aim and objectives

Outcome analysis of the occurrence of cardiovascular adverse events and cardiovascular risk in RA patients on treatment with JAK inhibitors.



Material and methods

Observational, retrospective, multicentre study of all patients with RA treated with JAK inhibitors for a minimum duration of 12 months.
Clinical variables: sex, age, smoking, Charlson Comorbidity Index, cardiovascular risk score, previous cardiovascular pathologies, Jak inhibitor treatment, duration, adverse effects or dose modifications.

Major adverse cardiac events (MACE) were recorded and when they occurred as well as their outcome (emergency visit, hospitalization, death).
Data was obtained from oncology electronic prescription and electronic medical records.
R commander® was used for the statistical analysis.

Results

71 patients with JAK inhibitors treatment

Median age	58 (IQR 48.5-65.5).	
Sex	85.9% female	
Charlson Comorbidity Index	1 (IQR 1-2	
Smokers	18.3%	
Median 10-year risk of cardiovascular events	1.5%b(IQR 1-3)	
Comorbidities at the start of treatment	arterial hypertension	32.4%
	dyslipemia	25.3%
	Diabetes	11.2%
	Ischemic heart diseases	4.2%
	Cerebrovascular disease	4.2%
Treatment	• 38 patients with tofacitinib. • 17 patients with baricitinib. • 12 patients with upadacitinib. • 4 patients with filgotinib. • 42% discontinued treatment (25% remained in therapeutic abstinence)	
Median treatment duration	31 months (IQR 13.1-40.9).	
MACE	12 patients (16.9%) developed some MACE during the course of treatment (6 dyslipemia, 3 arterial hypertension, 1 diabetes, 1 ischemic heart disease and 1 deep vein thrombosis) . Two patients needed hospitalization.	
Median year of treatment at which MACE developed	second year (IQR 1-3).	
After discontinuation	After discontinuation of treatment, 2 patients developed MACE: atrial fibrillation one year after discontinuation and pulmonary thromboembolism 4 years later. No patient died of MACE.	

12.6% reduced the dose, 7% increased dosage and 54.9% discontinued treatment (48.7% secondary failure, 23% adverse effects, 17.9% primary failure and 10.2% other reasons).
56.4% of patients who discontinued started treatment with another Jak inhibitor. The most prescribed treatment was upadacitinib in up to 50% of the cases.

Statistically significant differences were found between the occurrence of MACE and smoking habit (p=0.01)

Conclusions and relevance

The development of MACE occurs in a modest number of patients with no associated mortality in this study. A statistically significant association was found with smoking habit. It is necessary monitoring and management of modifiable risk factors in this patient population.