Medical or Research Professionals/Clinicans

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ACUTE MYOCARDIAL INFARCTION IS DRIVEN BY CHRONIC SYSTEMIC INFLAMMATION, IRRESPECTIVE OF THE KIND OF TREATMENT - DATA FROM THE GERMAN BIOLOGICS REGISTER RABBIT

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Background: Rheumatoid arthritis is associated with increased cardiovascular morbidity and mortality including acute myocardial infarction (AMI). Chronic systemic inflammation and accelerated atherosclerosis contribute to this risk. In daily practice, RA-patients with higher disease activity are more likely to receive treatment with biologic agents. However, there is insufficient evidence whether and how these drugs influence CV-risk.
Objectives: We aimed to evaluate the impact of treatment with biologic and non-biologic disease modifying anti-rheumatic drugs (bDMARDs, nbDMARDs) on the risk of AMI.
Methods: Nested case-control study with patients enrolled in the German biologics register RABBIT between May 2001 and October 2011. Cases were patients who developed an AMI during follow-up. Diagnoses of AMI were re-validated at on-site visits. Medical records as well as hospital reports were analysed. The availability of 9597 control patients without AMI enabled us to apply an comprehensive matching algorithm. In addition to matching for age, sex and year of enrolment, agreement was ensured for the following prevalent co-morbid conditions: heart failure, other coronary heart disease, hypertension, hyperlipoproteinemia and cerebrovascular disease. Regarding the exposure to biologic agents we applied a 3-month risk window (9 months for rituximab). McNemar test and paired t-tests were applied.
Results: In 68 patients AMI was re-confirmed. We found considerably more controls (N = 46) exposed to any bDMARD than cases (N = 32), although the difference was statistically not significant (p=0.07). The disease activity score (DAS28), use of glucocorticoids or functional capacity were not associated with the risk of AMI. Statistically significant predictors of AMI were elevated erytrocyte sedimentation rate (ESR, p<0.001) as well as C-reactive protein (CRP, p=0.03).

	Cases	Controls	p
Female n (%)	41 (60.3)	41 (60.3)	*
Age mean (SD)	63.8 (9.4)	63.8 (9.4)	*
ESR mm/h (mean over time)	37.1 (24.8)	25.5 (15.2)	<0.01
CRP mg/l (mean over time)	20.8 (21.1)	13.6 (16.4)	0.03
DAS28 (mean over time)	4.6 (1.2)	4.5 (1.2)	0.49
Any biologic DMARD n (%)	36 (52.9)	46 (67.7)	0.07

* matching criteria
Conclusions: Using data from a large observational cohort, we confirmed the importance of chronic inflammation for the risk of AMI. Whereas acute phase reactants were significantly associated, the composite measure DAS28 was not. The biologic link of AMI with measures of systemic inflammation may be closer than with signs and symptoms of joint inflammation (including also pain and patient global assessment). In addition, due to the matching procedure, chronic comorbid conditions were highly prevalent in this sample which may have contributed to the higher DAS28 in the nested case-control study than in the entire cohort. The kind of treatment (biologic or non-biologic DMARDs) did not have an influence on the risk of AMI beyond their influence on controlling inflammation.

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