PREDICTORS OF HEART RATE RECOVERY FOLLOWING EXERCISE IN PATIENTS WITH RHEUMATOID ARTHRITIS

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Background: Cardiovascular disease (CVD) is a major cause of death in rheumatoid arthritis patients (RA). The underlying mechanisms for the CVD increase risk in RA are not fully understood. However, poor parasympathetic function, a risk factor for cardiac mortality in the general population, has been implicated in RA. Little is known about the predictors of parasympathetic function in RA, which is most commonly assessed by heart rate recovery following maximal exercise testing.

Objectives: To explore the association of multiple factors including traditional CVD risk factors, global CVD risk score, VO2 max, quality of life, and inflammatory markers with heart rate recovery (ΔHRR) following an exercise tolerance test, a marker of parasympathetic function.

Methods: 106 RA (54.5 ± 12.3 years, 68% women) patients completed an exercise tolerance test on a treadmill, during which heart rate (HR) was monitored via 12 lead ECG. ΔHRR was quantified as the difference between maximal HR during exercise and HR one minute post exercise. Cardiorespiratory fitness (VO2 max) was assessed via breath by breath gas analyses. A fasted blood sample was taken to examine levels of C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), fibrinogen and white blood cells (WBC), lipids profile, and markers of insulin sensitivity (HOMA, QUICKI). Quality of life was assessed using the EuroQol questionnaire. The Framingham Risk Score (FRS) was used as an assessment of global risk for CVD.

Results: The average drop in HRR was 29 ± 13 beats per minute. Partial correlation (controlling for gender) analyses revealed that age (r = -0.25, p =0.03), resting HR (r = -0.34, p =0.003), triglycerides (r = -0.25, p =0.03), fibrinogen (r = -0.33, p =0.004), ESR (r = -0.24, p =0.03), WBC (r = -0.26, p =0.02), FRS (r = -0.28, p =0.01) were inversely associated with ΔHRR, whereas EuroQol (r = 0.26, p =0.02) and VO2 max (r = 0.28, p =0.01) were positively associated with ΔHRR. To identify independent predictors of parasympathetic activity in RA, stepwise linear regression was conducted. This revealed that fibrinogen (p =0.03), resting HR (p =0.001), FRS (p = 0.009) and EuroQol (p = 0.01) were independently associated with ΔHRR.

Conclusions: Even though several individual CVD risk factors, such as inflammatory markers, fitness level, and quality of life were associated with parasympathetic activity, only fibrinogen, global CVD risk and quality of life were independently associated with parasympathetic activity in RA. These findings suggest that parasympathetic reactivation is predicted by overall CVD risk rather than individual risk factors. Longitudinal studies should
explore how a reduction in CVD risk factors - achieved via exercise – may impact on parasympathetic activity in RA.

Disclosure of Interest: None declared