The undiagnosed, untreated key to restoring physical function and health in rheumatoid arthritis patients

Rheumatoid arthritis (RA) is characterised by adverse changes in body composition – specifically, reduced muscle mass (MM) and increased fat mass (FM). Indeed, when body composition is assessed (i.e. MM and FM are estimated), significant muscle loss is evident in ~67% and obesity in ~80% of RA patients. The loss of MM (and subsequent loss of strength) and extra FM (increased load) contribute to impaired physical function (disability), a feature of RA. Whilst obesity, particularly trunk obesity (fat is preferentially deposited around the trunk in RA) exacerbates the already elevated risk of developing cardiovascular disease (CVD) and diabetes.

Unfortunately, these body composition changes, termed ‘rheumatoid cachexia’ (RC), are rarely noted in clinics as patient’s bodyweight and body mass index (BMI) usually remain stable and normal, and body composition is not assessed. As RC is not detected, it is rarely treated.

Current treatments of RA (generally adopted ~2008) have as their central theme ‘tight control of inflammation’ by disease-modifying anti-rheumatic drugs (DMARDs). This approach emphasises early and aggressive use of DMARDs with an increase of dose, addition of drugs, or change of drug if the treatment goal (‘remission’ or low disease activity) is not achieved.

Since RC is driven by inflammation, one would anticipate that the success of stringent drug strategies in controlling inflammation would reduce RC and hence attenuate disability and co-morbidity risk in RA patients. Unfortunately, this is not the case. In a recent study we compared 82 RA patients, all exclusively treated by current ‘tight control’ drug strategies, to healthy sedentary individuals of the same age and sex. Whilst the treatment of RA patients had been clinically effective (half had achieved ‘remission’ with the remainder generally having ‘low disease activity’; categories determined by measures of inflammation), the magnitude of muscle loss and obesity, especially trunk obesity, was the same as before the adoption of ‘tight control of inflammation’ strategies. Consistent with the absence of improvement in body composition, fitness tests designed to reflect the ability to perform daily tasks (getting dressed, doing housework, carrying shopping etc.), consistently showed that patients’ physical function was only two-thirds of that of sedentary healthy individuals matched for age and sex. Put another way, the RA patients, whose average age was 60 years, typically had the function of healthy individual’s aged 85 years! Additionally, since adiposity, particularly trunk FM, was not reduced, patients’ CVD and diabetes risk remained elevated.

Given that RC is inflammation-driven, why does ‘tight control’ drug treatment not reduce RC? A likely explanation is that RC primarily occurs before RA is diagnosed and DMARD treatment commenced. Thus, DMARD treatment starts too late to prevent it and, since none of the drugs used are anabolic (i.e. increases MM), fails to restore body composition or, as a consequence, normal levels of physical function or co-morbidity risk.

So what can be done by health professional to resolve this situation? Firstly, body composition needs to be assessed in RA patients at diagnosis and at least annually thereafter [bioelectrical impedance is a quick, simple and inexpensive method suitable for clinics]. Such assessment would alert physicians to the need to treat RC, and indicate when anabolic therapies are optimally commenced. As to therapies that specifically aim to improve body composition and thereby restore function and co-morbidity risk; clearly the most effective in RA patients, as for the general population, is high-intensity exercise, particularly resistance training. Thus, appropriate exercise programs should be recommended to patients at diagnosis.

Recognition of RC and appropriate adjunct treatments to reverse it are required if meaningful improvements in patients' disability and well-being are to be achieved.

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