education, and higher fatigue, whereas older age and greater pain were associ-ated with persistent disease activity in men. Smoking cessation in men and weight reduction in women, and optimising MTX use may facilitate rapid reduction of inflammation, an essential goal of treatment in early RA.


Disclosure of Interest: None declared


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Fat and fatty acids: targets for therapy?

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Background: Adipocytokines are implicated in the development of fibrosis, vasculopathy and immune abnormalities through a variety of biological effects, but their role in systemic sclerosis (SSc) is not fully investigated. Chemerin is implicated in chemotaxis of immune cells, in promoting angiogenesis and it is involved in inflammation. Adiponectin (APN) has metabolic actions and anti-inflammatory properties, while Leptin (LEP) mediates actions in endothelial cells, such as angiogenesis, vasodilation, NO production and upregulates various mediators of vascular inflammation.

Objectives: In this study we investigated Chemerin, LEP and APN levels in SSc patients according to disease subtypes and clinical characteristics.

Methods: Chemerin, LEP and APN levels were evaluated in 100 SSc patients and in sex, age and BMI matched healthy controls. Clinical and demographic characteristics were available for all patients.

Results: Chemerin, APN and LEP levels were lower in SSc patients compared to healthy controls (Chemerin: 58.7±27.6 ng/ml vs 74.0±29.0 ng/ml, p=0.004; LEP:19.6±18.3 ng/ml vs 28.5±23.8 ng/ml, p=0.03, APN: 6.5±3.9 µg/ml vs 12±6.0 µg/ml, p=0.001)

Chemerin levels were lower in patients with anti-topoisoemerase antibodies (50.2±22.7 ng/ml) respect to patients with other autoantibodies (64.6±29.7 ng/ml), p=0.018.

Regarding capillaroscopic damage, Chemerin levels were lower in patients with late pattern (44.8±18.9 ng/ml) compared to patients with early (64.3±28.5 ng/ml) and active pattern (71.7±29.9 ng/ml), p<0.001.

Chemerin levels inversely correlated with IL-6 levels (R=0.5, p<0.01), while directly correlate with capillary density (R=0.5, p<0.01). Chemerin levels also inversely correlated with PAPs on echocardiography (R=-0.24, p=0.02). Finally LEP levels inversely correlate with liver score or serum leptin level was low, even though leptin was significantly induced by HFD. However, DMM decreased leptin levels at all time points, independent from diet (e.g. 4 weeks: HFD healthy 18.4 ng/ml vs HFD DMM 3.7 ng/ml). The different parameters for metabolite changes (fatty liver score and bodyweight) were positively correlated with serum leptin level. HFD, DMM or the combination of both did not show significant effects on serum levels of adiponectin, leptin and visfatin and inflammatory markers. Diet induced systemic changes were also analysed using a fatty liver score and evaluation of crown-like structures (CLS) in adipose tissue. OA progression was scored and quantified based on histological stainings of the joints (H/E, safranin O). Immunohistochemical stainings of the joints were performed to evaluate local distribution of adipokine positive cells and the respective cell types. Metabolic parameters were correlated to the local progression of OA.

Results: The numbers of CLS were significantly lower comparing HFD (0.2±0.1553, n=7) with ND (5.219±0.9831, n=8) in mice. Fatty liver score was significantly higher in HFD compared to ND. OA induction was significant at every time point vs healthy control and higher in HFD (e.g.: OA score at 6 weeks HFD 3.7 vs ND 1.4). In the OA group, there was a positive correlation between bodyweight and OA score (r²=0.33). Correlation level of OA score with liver score or serum leptin level was low, even though leptin was significantly induced by HFD. However, DMM decreased leptin levels at all time points, independent from diet (e.g. 4 weeks: HFD healthy 18.4 ng/ml vs HFD DMM 3.7 ng/ml). The different parameters for metabolic changes (fatty liver score and bodyweight) were positively correlated with serum leptin level. HFD, DMM or the combination of both did not show significant effects on serum levels of adiponectin, leptin and visfatin or IL-6. Local adipokine secretion in the joints was independent from systemic metabolism parameters.

Conclusions: Our data show that similar to observations in humans, OA is deteriorated by HFD which correlates mainly with the bodyweight and to a lower extend with metabolic changes induced by obesity. Local adipokine expression was especially detectable in the damaged menisci showing increased amounts of adiponectin and leptin producing cells. Interestingly, local adipokine expression was independent from systemic adipokine levels.

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Biologics in RA. Improving and maintaining the response.

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Background: B-cell depletion is a fundamental effect of rituximab (RTX). The speed/depth of initial B-cell depletion is associated with clinical response, and non-responders largely having incomplete depletion. However, some patients with incomplete depletion still show clinical improvement (ID-R). Little is known about factors associated with complete depletion; the long-term outcome of the two responder groups according to their level of depletion has not been studied yet.

Objectives: To assess factors that are associated with complete depletion and clinical response (CD-R) and compare the 3 year RTX retention between the two RTX responder groups (CD-R vs ID-R), with a view to inform practice on the optimal use of RTX in RA.

Methods: A prospective observational study was conducted in patients with RA who were treated with RTX in Leeds. Each initial cycle of RTX consisted of 2 x 1000 mg infusions, repeated either on clinical relapse or fixed 6 monthly

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