Faecal microbiota transplantation for Clostridium difficile infection resulting in a decrease in psoriatic arthritis disease activity

Sirs,

We describe a case of a woman with psoriatic arthritis, who after receiving faecal microbiota transplantation (FMT) for Clostridium difficile infection was able to cease her disease-modifying anti-rheumatic drugs (DMARDs) with sustained remission of her rheumatic symptoms. Therapeutic alteration of the gut microbiome with FMT has been demonstrated to be an effective therapy for Clostridium difficile infection and ulcerative colitis, with trials underway in many other diseases (1). Changes in the composition of gut microbiota, termed dysbiosis, have already been implicated in gut diseases such as Clostridium difficile infection (2). There are emerging data to support to role of dysbiosis in the pathogenesis of rheumatic disease (3), thereby raising the possibility of therapeutic alteration of the gut microbiome having an impact on disease activity in psoriatic arthritis.

A 59-year-old woman with a history of psoriatic arthritis presented to our institution in July 2016 with diarrhoea of up to twenty motions per day having recently completed a course of oral clindamycin for presumed facial cellulitis. The patient had been diagnosed with psoriatic arthritis in 2004 with recurrently active spondyloarthopathy and polyarthralgia, manifested as sacroiliac joint pain and stiffness, and synovitis of the small joints of the feet, left ankle and right knee. Previous whole body bone scan had demonstrated sacroilitis, although plain x-ray did not demonstrate erosions and an MRI was never performed. Mild psoriasis was her only extra-articular manifestation and had not been present for many years. In the 3 months preceding presentation, she complained of increased duration of morning stiffness to between 2-3 hours and increased pain in multiple joints including the sacroiliac joints. Clinically she had a left ankle effusion, a small-moderate right knee effusion, synovitis in left second to fourth metatarsal phalangeal joints and left sided plantar fasciitis. Tender and swollen joint count (TSJC) of 10 and CRP of 6.3mg/L. The return of symptoms coincided with an episode of traveller’s diarrhoea. The patient was reluctant to restart immunosuppression due to a fear of infection, so a compromise of oral methotrexate at 10mg/week was commenced with partial response. With regards to extra-articular disease, the patient had not had active psoriasis for over 24 months before the FMT, and there was no recurrence after the FMT, so we cannot comment on the effect of FMT on psoriatic skin disease in this patient.

To our knowledge, this is the first case report of FMT being associated with attainment of minimal disease activity in psoriatic arthritis.

Studies using 16s RNA sequencing have found significant differences in the both the gut and cutaneous microbiota of psoriasis patients compared to controls (6). Patients with psoriatic arthritis have been noted to have low levels of Akkermansia muciniphila species and Ruminococcus genera compared to controls (6). These are bacteria that have anti-inflammatory properties and are known to contribute to gut homeostasis (7).

The gut microbiota can influence local and systemic immune responses via alterations in intestinal permeability, molecular mimicry and activation of effector immune cells (8). Psoriatic arthritis is known to be a T cell-mediated disease, in which pathogenic T cells produce IL-17 in response to IL-23. This is supported by the fact that IL-17 producing T cells (Th17) are found in affected tissues (9) as well by the fact that agents that target the IL-23/Th17 pathway (e.g. ustekinumab) appear to be efficacious. In a mouse model, segmented filamentous bacteria induced autoimmune arthritis through the ability to specifically promote differentiation of the Th17 subset (10). It is therefore plausible that gut dysbiosis may directly contribute to the direction of dif-

Letters to the Editors
Diferentiation of naïve T cells into specific effector T cells that drive the pathogenesis of psoriatic arthritis.

The outcome in this case is hypothesis-generating in that it suggests that the gut microbiome may play an aetiological role in psoriatic arthritis and there may be the potential to treat psoriatic arthritis with gut microbial manipulation. Clinical trials aimed at modulating the gut microbiome using FMT or defined microbial consortia are required to further investigate these possibilities.

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