
Lymphatic Function in Inflammatory Arthritis

Sir—We read with interest the case, reported in a letter by McRorie et al. [1], of a woman with congenital lower limb lymphatic hypoplasia in whom the development of RA resulted in the onset of lymphoedema in one leg and the exacerbation of lymphoedema in the other leg.

We have compared lymphatic drainage in the upper limbs of patients suffering from inflammatory arthritis with and without co-existent lymphoedema and found an impairment of lymph drainage only in the oedematous group [2]. As there was no reduction in lymph drainage in the patients with inflammatory arthritis, and no oedema, we conclude that inflammatory arthritis alone does not directly impair lymph drainage. We have therefore proposed that in patients with inflammatory arthritis and lymphoedema, there must be an additional unrelated factor impairing lymphatic function which primarily places them at risk of developing lymphoedema [2]. The case described by McRorie et al. [1] illustrates and supports this hypothesis well: the onset of RA resulted in a critical increase in local capillary filtration to an extent that could not be accommodated by the hypoplastic abnormal lymphatics, resulting in the onset of oedema in one leg and its exacerbation in the other.

Unfortunately, in the clinic, it is not possible to predict which patients with inflammatory arthritis are at risk of developing lymphoedema, unless lymphatic function is already known to be abnormal. Unlike the case described by McRorie et al., others describe the persistence of oedema after the resolution of arthritis (reviewed in [3]), suggesting that a prolonged increase in load (i.e. increased capillary filtration) through abnormal lymph vessels may induce further damage and lead to permanent oedema. To avoid this, we suggest prompt anti-inflammatory treatment to reduce capillary filtration (and hence the load on the lymphatics) beneath the oedema-inducing threshold, thereby preventing further damage to lymphatics, reversing oedema and preserving function in the affected limb.

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Flatfeet in Pregnancy

Sir—We report two cases of flatfeet developing for the first time during pregnancy; this caused significant and lasting disability in one patient.

A 35-yr-old woman presented in the third trimester of her second pregnancy with an acute onset of flatfeet. Her weight gain was ~3 stones, she had marked peripheral oedema and consequently had walked barefoot as she had found shoes uncomfortable. Her main symptoms were pain along the medial aspect of her feet on weightbearing and ill-fitting shoes (her shoe size increased from 39 to 41). Her symptoms were so severe that she had to use a wheelchair for several weeks. The patient had had several miscarriages (all more than a year before this pregnancy) and hyperemesis in her first pregnancy. Her cousin had also had flatfeet in pregnancy. At the time of writing, her pain had subsided into an intermittent ache and although her shoe size had returned to normal, she was only comfortable in supportive shoes, such as trainers.

On examination (9 months post-partum), she had significant loss of the medial arch of her foot. The rest of her joints were normal, with no hypermobility. Her pain slowly improved with physiotherapy, insoles and wearing supportive shoes; however, her flatfeet still persist. An MRI showed normal tibialis posterior tendons.

Our first patient put us in touch with a 38-yr-old woman who had developed pain in her feet on weightbearing at 30 weeks gestation. Her weight gain was over 4 stones. After delivery, she noticed that the shape of her feet had changed and she was no longer able to wear her old shoes. Her symptoms gradually improved, although 3 yr later her feet were not back to normal. This was her first successful pregnancy: she had had numerous miscarriages (ranging from 6 to 26 weeks gestation), cause unknown. Her most recent had occurred 3 months prior to this pregnancy and had been a 20-week twin pregnancy. She had suffered long-standing back ache and in 1981 had had bilateral osteotomies for bunions. On examination (3 yr post-partum), she had bilateral flatfeet and no evidence of joint laxity.

A Medline search from 1966 to the present day did not reveal any case reports of flatfeet in pregnancy. However, the fact that our first patient knew two other cases personally suggests that the condition may be quite common. The problem may often be so minor
as to not cause concern and this may explain why there have not been any cases reported in medical journals.

Flatfeet are known to occur in conditions with hyperlaxity, such as the rare Ehlers-Danlos syndrome [1]. In such conditions, flatfeet are known to deteriorate during pregnancy [2]. There are several reasons why flatfeet may occur in pregnancy: significant weight gain and increased ligamentous laxity due to the hormonal changes will both play a part. Relaxin is a peptide hormone with a structure not dissimilar to insulin. There is evidence that it is produced by the corpus luteum in pregnant women and has important hormonal functions, including a direct effect on collagen [3, 4]. It could be postulated, in view of the fact that both are produced in pregnancy, that relaxin could be postulated to have a similar function. Perhaps pregnant women should be given advice regarding supportive footwear and avoid going barefoot in the hope that this could prevent the development of flatfeet. In the future, it may be appropriate to carry out a prospective study to gain further information on the incidence and severity of foot problems during pregnancy.

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CD69 on Synovial T Cells in Rheumatoid Arthritis Correlates with Disease Activity

Sir—A number of activation markers have been described on the surface of synovial fluid T cells which have different functions. CD69 is a very early T-cell activation marker and up to 70% of synovial T cells express it [1]. We have recently presented evidence to show that its upregulation occurs at the time that T cells enter the inflammatory focus [1]. The particular importance of CD69 is that it is involved in the direct stimulation of macrophages by activated T cells to secrete inflammatory mediators such as interleukin-1 [2]. We reasoned that the number of CD69 T cells in the synovial fluids of patients with rheumatoid arthritis (RA) should have some relationship to the degree of joint inflammation.

The peripheral blood and paired synovial fluid T cells from 10 patients (five males, five females; age 54.5 ± 12.7 (s.d.) yr), fulfilling the ARA criteria for RA, were examined by cytofluorographic analysis using the FACScan (Becton-Dickinson). As expected, the percentage of CD69-positive T cells was low in the peripheral blood (3.2 ± 2.0 s.d.), while there was a significant increase in the synovial fluid (65.4 ± 19.0 s.d.; $P < 0.001$). Similar results were obtained for the surface expression of CD69 (mean fluorescence intensity: for blood, 1.7 ± 1.0 s.d.; for synovial fluid, 32.1 ± 13.0 s.d.; $P < 0.001$). Of interest was the finding that both the percentage of CD69 and the mean fluorescence intensity correlated with the number of inflamed joints (Fig. 1; $r = 0.76$, $P = 0.04$ and $r = 0.81$, $P = 0.03$, respectively).

From these results and the in vitro data of Dayer and colleagues [2], it may be concluded that inhibition of the actions of CD69 could suppress disease activity in RA.

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