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Lymphoedema management in podoconiosis

As researchers in the skin treatment of podoconiosis, we were interested to read the Article by Henok Negussie and colleagues on lymphoedema management to prevent acute dermatolymphangioadenitis in podoconiosis (July, 2018).¹

We were, however, concerned about the interpretation of our cited trial on skin barrier function in those with podoconiosis in Ethiopia.² Negussie and colleagues state that our trial was a "small study". In fact, it comprised 193 participants with podoconiosis. This number was established as follows: to achieve a power of 80% (two-sided t-test) with a 5% significance level, 64 participants were required, divided equally between the two treatment groups. To allow detection of possible smaller effects on other secondary outcome measures, a target sample size of 200 was chosen.

Negussie and colleagues also state that "small volumes of water with glycerol showed equivalent effects on skin barrier function to treatment with larger volumes of water". This was not the case. At all three points on the lower outer lea and on the top of the foot, measures of transepidermal water loss and stratum corneum hydration using 1 L of the 2% glycerine water soak (experimental group) compared with the 6 L water soak (control) indicated a statistically significant improvement over the 3-month trial. For example, on top of the foot, the estimated group difference in transepidermal water loss at the fourth visit was 1.751 (SE 0.0390) in favour of the experimental group (t=3.154; degrees of freedom [DF] 189.580; p=0.002), indicating a greater reduction in transepidermal water loss in the experimental group. Similarly, at the same site, the estimated group difference in stratum corneum hydration at the fourth visit was -2.041 (SE 0.572) in favour of the experimental group (t=-3.565; DF 186.739, p<0.001), indicating a greater increase in stratum corneum hydration in the experimental group.

Furthermore, Negussie and colleagues state "to our knowledge, GoLBeT is the first trial to assess the effects of a lymphoedema treatment package on the most important clinical consequence of lymphoedema: the incidence of acute dermatolymphangioadenitis". Our randomised controlled trial recorded work days lost due to adenolymphangitis (defined as an episode of inflammatory pain associated with the lymph nodes within a lymphoedematous leq which led patients to be bedridden or unable to work). At baseline, the mean number of work days lost in the previous month due to acute dermatolymphangioadenitis was 4.56 for the control group and 4.44 for the experimental group. At visit four, however, no participants in either group had lost any work days in the previous month due to acute dermatolymphangioadenitis.

Additionally, we propose that acute dermatolymphangioadenitis is only a surrogate of the primary failure point—stratum corneum barrier function—as one can address this profound systemic condition simply with topical treatment of water, soap, and petrolatum.

We declare no competing interests.

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2

