The skin as a barrier: What does it mean when it fails when lymphoedema is present?

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The skin is a barrier and a primitive immunosurveillance organ. When in good health at rest, the skin has no reason to undergo repair. Stretching, compression or stimuli such as stripping with tape or exposure to UVB switch on the repair mode (Ryan, 2004). After a short period, often measured in hours, mitoses appear and cytokine production stimulates the dermis into activity. The resulting inflammation heats the tissue and recruits white cells.

It is the barrier function of the skin which most often fails in lymphoedema, irrespective of the cause of the lymphoedema. For some patients in the later stages of lymphoedema, the disfigurement impairs social interaction and this can mean they are unwelcome in some communities. In podoconiosis, which is common in Africa, the lymphoedema occurs primarily in the foot and lower leg. Here it is due to barefoot exposure to an irritant soil. It is most common in Ethiopia.

One outcome of the loss of barrier function is loss of control of transepidermal water loss which can lead to desiccation. At a basic level, effective treatment under these circumstances can be as simple as provision of shoes, washing the affected areas, and emollients (Ferguson et al, 2013).

It is worth noting that most of the therapy for lymphoedema, irrespective of the cause, is directed at the skin. The intactness of the barrier depends on moisturising. Getting the balance right is critical since too much can mean maceration. On the other hand a dry epidermis cracks and allows entry of bacteria and soil irritants, destroying the superficial lymphatics, leading to lymphatic insufficiency of the foot and lower leg. In many countries (especially developing ones), fissuring of the skin is alarmingly common in agricultural workers who do not wear shoes.

Most patients with lymphoedema tend to keep the limb immobile. While this might initially be the most comfortable option, it does result in the lymphatic system (especially if it is damaged) having difficulty removing fluid, and thus there is further limb swelling. The lack of mobility and gravitational effects on the venous system also result in secondary venous failure. So we have two systems failing! Subsequent ulceration, a severe loss of the skin's barrier function sometimes seen in the lymphoedematous limb, is almost always due to venous failure. The impaired oxygenation keeps the epidermis in poor health and the skin suffers from a patchy distribution of a range from dry and brittle to wet and severe oozing well beyond the ulcerated area.

Thus the "repair mode" does more harm than good when it is prolonged and continuous. In order to switch off the repair mode, most often all the affected skin needs, whether eroded, or red and scaly, is a an emollient applied to the surface to seal its cracks and moisturise the desiccated cells – a simple requirement ignored commonly in both developed and developing countries.

As an example, the Institute of Applied Dermatology in Kasargod, Kerala in India applies therapy that includes soaking in a herbal mixture (the exact role of which has not yet been studied) with allopathic investigative procedures (Ryan and Narahari, 2012). This mixture is likely to be antiseptic and it may suppress activation of cytokines that play a role in the inflammatory state which is a feature of elephantiasis. If these can be inhibited it is the first stage in the normalisation process.

Skin care is not the total picture, however. It is the Indian use of yoga that counteracts immobility and gravitational effects which enhance swelling of the affected area, and often the whole limb. The emptying of the great veins and lymphatics in the chest through breathing effectively and changing intra-thoracic and intra-abdominal pressures would seem to be especially important (Piller et al, 2006), which hardly ever happens in the immobile patient. Already we are seeing objective evidence relating to the benefit of yoga in lymphoedemas (Louden et al, 2012).

In contrast to what might seem reasonable or logical, heating has a long-established role in Asian therapies for lymphoedema, although the objective evidence is yet to be collected. It is known that as long as there is no infection that a lymphoedematous limb is well below the optimum temperature of 37°C and there are good reasons to use heating as a therapeutic tool, but what temperature and for how long remains to be elucidated. Molloy et al (1993) and Molloy and Egger (2008) argued that prolonged overheating, especially in the home, was bad for the skin. Certainly cooling from evaporation of surface trans-epidermal water and from erosions and ulcers might be an issue. Also if a dressing changes take a long time, considerable cooling can occur and it may take many hours to recover from suboptimal temperatures to allow the processes for repair to restart.

So where does the balance point lie? Certainly protecting the skin as a barrier is critical. Just as important seems to be mobility and activity, and active rather than passive breathing. Maybe we need to think more about the effect of limb temperatures. Some of these thoughts might seem counterintuitive, but we still need to consider them and collect evidence. It is clear that for lower limb lymphoedemas we need to look for strategies which help both the lymphatic and vascular systems.

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