Tissue viability in rheumatoid arthritis

Jill Firth
Smith & Nephew Foundation Doctoral Student, University of Leeds, and
Honorary Rheumatology Nurse Specialist, Bradford Teaching Hospitals NHS Foundation Trust

Key words: tissue viablility, rheumatoid arthritis, risk factors, ulcers, wound healing

Introduction

Rheumatoid arthritis (RA) is the most common form of inflammatory arthritis with an estimated prevalence from pooled studies of between 0.5 and 1.0 per cent of the population[1]. Tissue viability is compromised in RA for a number of reasons including manifestations of the disease process, reduced mobility and self-care capacity. This manifests itself in increased prevalence of pressure ulcers, lower limb ulceration and impaired wound healing.

This article outlines factors that contribute to impaired tissue viability in RA and describes the clinical manifestations that occur as a result. The literature cited in this review is taken from a series of literature searches that form the basis of the author's doctoral studies.

Factors affecting tissue viability in rheumatoid arthritis

So what is it about RA that forms the association with an increased prevalence of wounds and delayed healing? The clinical picture is complex and dependent to some extent on the severity of disease and the effects on the individual. The following factors, which are summarised in Table 1, need evaluation in order to consider the promotion and maintenance of tissue viability by the multidisciplinary team.

Cutaneous manifestations of vasculitis

The clinical spectrum of rheumatoid vasculitis ranges from inflammation of the venules and small arteries to a necrotising arteritis that may affect small and medium sized arteries leading to wider organ involvement.

The vasculitis most closely associated with RA is a leukocytoclastic vasculitis involving post-capillary venules in which the skin is the organ most likely to be affected[2,3]. It is the involvement of the small vessels that may manifest itself as palpable purpura, erythematous nodules or ulceration and necrosis in the lower leg and foot[3,4].

The ulcers typical of vasculitis usually differ in both appearance and site from those associated with direct pressure. Common sites are the lower leg and the dorsum of the foot, although skin lesions can appear on areas of local pressure and previously traumatized skin[3,5].

In contrast to pressure ulcers, vasculitic ulcers may appear suddenly, frequently preceded by palpable purpura and ecchymosis. Lesions appear well demarcated, of variable depth and size, and are often painful and slow to heal[4].

Leg ulcers in RA are often attributed to vasculitis although there is little evidence for this as few studies have critically evaluated the role of individual risk factors in ulceration[6,7]. Those which have, albeit in studies with small sample sizes, conclude that the aetiology of the ulcers was multifactorial, with venous and arterial insufficiency, trauma or pressure to be factors commonly involved[8-10].

Rheumatoid vasculitis should be considered when a patient presents with cutaneous ulceration, particularly if other extra-articular manifestations of disease are evident but other possible risk factors should be given equal weight.

Nodules

Rheumatoid nodules have been found to affect 15-39 per cent of patients in studies of patients with systemic disease[11].

Nodules tend to occur over bony prominences where the skin and subcutaneous tissues are already under increased stress. They vary in size and number and may persist indefinitely or can regress at the time.

Common sites include the elbows, heels and sacrum. Nodules are a possible risk factor for ulceration as they have the potential to increase pressure and friction from extrinsic factors as well as affecting local tissue viability.

Anaemia

A systematic review of the prevalence of anaemia in RA found estimates to range between 33 per cent and 60 per cent, with most of the cases being characterised as the anaemia of chronic disease[12]. Anaemia, whether normochromic, normocytic due to disease activity or iron deficiency as a result of drug-induced gastric bleeding, reduces the quality of blood perfusing peripheral tissues impairing healing.

Decreased energy levels as a result of anaemia may also combine with fatigue and impaired mobility to further increase pressure risks to tissue viability. Added to this are the complex immunological changes that can be seen in the blood and tissues affecting phagocytosis and specific immunity–defence mechanisms[13,14].

Foot deformity

Figure 1 overleaf highlights aspects of foot deformity in RA that are areas at risk of ulceration. The lesser metatarsal heads herniate through the plantar capsule and the base of the proximal phalanges then lie on the dorsal aspect of the lesser toes[15,16].

The plantar fat pad is thus displaced and this results in prominent metatarsal heads just below the skin. This increases plantar pressure and callosities form at the site[17]. Displacement of the joints creates an imbalance between the intrinsic and extrinsic muscles that lead to the development of hammer or claw toe deformities. Contraction of the toes creates the potential for friction, pressure from footwear and abnormal load bearing, all of which contribute to callus formation.

The same process causes the great toe to gradually drift into a valgus deformity. This is described as hallux valgus, which is often associated with the development of a bunion over the medial aspect of the first metatarsal head[18]. The joints of the midfoot only have a narrow range of movement but flattening of the longitudinal arch and valgus heel deformity both occur in RA.

In valgus heel deformity, foot pressure studies have shown a shift in pressure from the lateral metatarsal heads to the medial forefoot, associated with a higher prevalence of callosities[19]. At the heel itself rheumatoid nodules may develop on the plantar surface of the calcaneus as a result of the pressure from ambulation and friction from footwear[16].

Foot deformity may cause a direct or indirect threat to tissue viability in the rheumatoid foot. Lavery et al (1998) found in a study of 76 diabetic patients and 149 case controls that 78 per cent of patients had a rigid deformity directly associated with the site of ulceration[20].

A small study by Mueller et al (1990) of 40 diabetic patients with ulceration also found a significant association between foot deformity and location of the ulcer ($p<0.001$)[21]. In addition, foot deformity is a risk

TABLE 1
Contributory factors to impaired tissue viability in RA

Reduced mobility and self care capacity	Increased pressure risks Difficulty with skin care
Poor nutrition	Poor appetite due to pain, inactivity, fatigue or depression Restricted jaw opening Sjogrens syndrome Inability to prepare food
Medication	Non-steroidal anti-inflammatory drugs Cytotoxics Anti-TNF therapies
Foot deformity	Raised plantar pressures Friction from ill-fitting footwear
Extra-articular manifestations of disease	Vasculitis Nodules Peripheral neuropathy
Peripheral vascular disease	Increased prevalence