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Editorial

Simple advice for a simple ankle sprain? The not so benign ankle injury

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Osteoarthritis (OA) is the most common joint disorder in the Western World, and is one of the most frequent causes of pain, loss of function and disability in adults. As the most prevalent load bearing sites for OA, much research has been conducted focussing on the knee, followed by the hip. However, comparatively little research has been conducted on the ankle. This may perhaps be due to the low prevalence of ankle OA, presenting around 4% of all OA cases, especially when compared with the knee and hip, presenting 41% and 19%, respectively[1].

In different joints, the natural history of OA appears to vary, with the factors contributing to disease occurrence and progression appearing to be joint specific[2]. Female sex has been shown to amplify the age-related increases in the risk of hand and knee and multiple joint OA (so-called 'generalised OA'), so that after the age of 50 years the prevalence and incidence of disease in these joints is significantly greater in women than in men[3]. In addition, hip OA has been shown to progress more rapidly in women than in men but with no gender effect observed on the progression of knee OA[4,5]. There is a stronger association between obesity and speed of OA progression at the knee, compared with the hip. Again while much is known about the joint specific risk factors for the knee and hip conversely very little is known about risk factors for ankle OA, and the influences factors such as gender, age and obesity have on its prevalence and progression.

The prevalence of different types of OA also differ by joint, with the ankle appearing 'resistant' to primary OA development with between 9% and 15% spontaneously occurring, compared with around 82% for knee OA[6]. Differences in biomechanical and mechanical properties of articular cartilage between the knee and ankle have been observed previously, with ankle cartilage being both thinner and stiffer than that of the knee[7]. It is thought that this type of cartilage is better adapted to distributing repeated stresses more evenly across the whole articular surface, compared with thicker and more varied distribution of cartilage within the knee, and hence is more protective of progressive, primary OA development.

While some joints (e.g., the ankle) may be more resistant to primary OA, there does appear to be a joint dependent response to stimuli such as injury, and it is suggested that the reaction of the ankle to trauma is different to that of other joints. Injury is a known risk factor for the development of OA, with 8% of hip OA being post-traumatic (due to injury), and 12% for the knee[8,9]. Worryingly, within the ankle, almost 80% of ankle OA is reported to be post-traumatic[6,10]. Valderrabano and colleagues studied the aetiology of ankle arthritis reporting 78% as post-traumatic OA (PTOA) compared with just 9% primary OA cases. Similarly, Saltzman *et al.* observed 70% of ankle OA was post-traumatic (primary 7%; rheumatoid 12%; other 11%), characterised largely as a result of an inciting event or insult. Acute injury to the joint may directly cause damage to cartilage and/or subchondral bone, influencing cartilage integrity and resulting in degenerative changes. Certainly, cartilage within the ankle is different to that of cartilage in other joints within the body, and in a recent animal study different joint articular cartilage responses to impact and injury were observed[11]. In addition to direct articular damage, damage to the joint surrounding structures may also lead to changes in lower limb biomechanics, resulting in uneven or altered distribution of load at sites that experience unaccustomed or excessively high loading. The resulting repeated abnormal joint loading, mal-alignment and instability is what is thought to lead to asymmetrical joint wear patterns, increased levels of ankle pain and the development of posttraumatic ankle OA[12].

Both Valderrabano, and Saltzman and colleagues observed that ankle arthritis patients with post-traumatic end-stage ankle OA were younger than patients with primary ankle OA (51 yrs vs 67 yrs, respectively). In addition, Schmal *et al.* (2014) reported that the age of individuals presenting with ankle related OA tended to be younger than individuals reporting other joint OA[13]. The latency between injury and the development of OA was also influenced by the severity of injury i.e., if there was a fracture, direct cartilage damage, and/or by the degree of ligament injury[10].

Ankle injuries are regularly reported as one of the most common sports related injuries, with around a quarter of injuries across all sports being ankle sprains[14]. Ankle injuries are also noted to be among the most common recurrent injuries within sport, with evidence indicating a two fold increase in risk of a second sprain for at least one year post injury, suggestive of ongoing dysfunction[15]. In the general population, ankle sprains are also very common, accounting for between 3% and 5% of all Emergency Department visits in the UK every year[16], but despite this they are often regarded as benign injuries that will resolve quickly with limited treatment or intervention. However, it is known that a large proportion of ankle sprains result in persistent debilitation, with the incidence of residual symptoms following acute ankle sprain reported between 30 and 50%[17,18].

While there is considerable evidence on the causation of ankle OA, and a much reported high prevalence of ankle injuries in

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both sport and the general population, there is a paucity of literature about a causal relationship between ankle injury and the development of post-traumatic ankle OA. What is it about those individuals who appear to do well following acute significant ankle ligament injury and recover fully, while others do badly? What is the likelihood of developing ankle OA after a significant ankle ligament injury, and what factors affect speed of onset and progression of the disease? Is the prevalence of post-traumatic ankle OA purely the result of an overall high occurrence of ankle injuries compared with other injuries to other body joints, and/or are adverse outcomes influenced by a lack of appropriate management and guidance post 'simple' ankle sprain?

As the aetiology of OA and in particular PTOA differs at different sites, there is a call for them to be treated as separate conditions. This lack of knowledge about the ankle specific risk factors for PTOA development and progression, and the influence of injury on joint integrity will have undoubted consequences for the prevention and management of ankle injuries and posttraumatic ankle OA.

With such an obvious link to the development of post-traumatic ankle OA, ankle sprains are rarely the simple and benign injuries they are often initially considered. Moreover, ankle OA should not be accepted as an inevitable consequence of ankle injury. In order to better treat ankle injuries, and combat and manage the long term consequences of injury such as ankle pain and dysfunction, including posttraumatic OA, we need to understand what happens between the occurrence of a significant ankle ligament injury, and end stage development of OA by conducting large prospective cohort studies.

Author contributions

All authors contributed to the development, writing and editing of the manuscript.

Conflict of interest

There is no conflict of interest for any of the authors.

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