The signs include alteration in joint shape, muscle atrophy, weakness, increased effusion, restricted movement, bony swelling and soft tissue swelling.

Pro-inflammatory cytokines are believed to play a pivotal role in the initiation and development of this disease process, among which IL-1ß and TNF- α appear prominent. Both IL-1ß and TNF- α increase expression of additional pro-inflammatory mediators, (prostaglandin E_2 (PEG_2), leukotriene B_4 (LTB_4), bradykinin, and nitric oxide), promote chemotaxis and degrandulation of leukocytes and increase the activity of several proteolytic enzymes that degrade articular cartilage, most notably the matrix metalloproteinases (MMPs) (Martel-Pelletier et al., 1999).

In the normal joint, cartilage and bone homeostasis are maintained through a balance of anabolic and catabolic processes, which are regulated by a number of mediators, in particular IL-1 (Carmona and Prades, 2009).

IL-1 is a cytokine with both pro-inflammatory and catabolic properties. It appears in two isoforms, IL-1α, which is constitutively present under normal healthy conditions, and IL-1ß, which is expressed under pathological conditions, such as osteoarthritis (Moldovan et al., 2000).

In osteoarthritis, excessive IL-1 production and activity tips the balance in favours of catabolic and anti-anabolic processes, resulting in a reduction in cartilage synthesis, an increase in cartilage degradation and subchondral bone remodelling. IL-1 also indirectly stimulates chondrocyte and synoviocyte apoptosis and plays a key role in inflammation, both of which contribute to the loss of cartilage (Martel-Pelletier et al., 1999).