characteristic erosion, found especially in PIP, is the so-called “saw tooth” appearance.

Results: Although the diagnosis of EOA is based on radiographic erosion, some clinical features may lead to a suspicion of EOA. According to the definition of “inflammatory OA”, abrupt onset of pain, swelling, redness, warmth and limited function of IP joints are common. Sometimes the same features may be observed in non-EOA cases, but usually at the disease onset, during the first year. Characteristic of EOA is the throbbing parasthesias of the fingertips. EOA may lead to joint deformities, some indistinguishable from those of non-EOA, such as lateral subluxations and Heberden’s and Bouchard’s nodes, while others are seen almost exclusively in EOA, such as instability and ankylosis of DIP and PIP and, rarely, opera-glass deformity.

Concerning laboratory investigations, ultrasonographic CRP has been proposed as marker of the disease activity. Some OA markers, such as the CTX I, were found to be increased in the serum and urine in EOA in comparison with nodal non-EOA. In another recent study, serum levels of myeloperoxidase (MPO) and, at lesser extent, Coll2−1NO2, were elevated in EOA in comparison with non-EOA.

Pattrick et al observed an increased frequency of the HLA-A1B8 and HLA-B7 alleles in patients with EOA. Among these, patients with EOA had an increased frequency of the MZ α1-antitrypsin phenotype (30 versus 9%). We found that HLA DRB1*0111 was associated with EOA, in comparison with non-EOA and reference populations. Stern et al reported an association between EOA and a genomic region containing the interleukin-1β (IL-1β)5810 single nucleotide polymorphism, thus supporting a potential role for IL-1 in the pathogenesis of this severe phenotype of hand OA.

In comparison with nodal OA, clinical aspects of EOA may sometimes be indistinguishable, although EOA is characterized by more frequent inflammatory episodes involving several joints simultaneously and may persist for many years, while nodal generalized OA exhibits its flares mainly at onset of the involvement of each joint, in a ‘stuttering’ onset polyarthropathy of DIPs and PIPs which resembles a ‘monoathritis multiplex’. Furthermore, instability and ankylosis of IPs are seen almost exclusively in EOA.

Conclusions: According to the recent EULAR recommendations for the diagnosis of hand OA, EOA may be considered “as subset of hand OA, characterized by radiographic erosions targeting IP joints which may progress to marked bone and cartilage attrition, instability and bony ankylosis. Typically it has an abrupt onset; marked pain and functional impairment; inflammatory symptoms and signs (stiffness, soft tissue swelling, erythema, paraesthesia); mildly elevated CRP; and a worse outcome than non-EOA”.


Conclusions: These studies show that OA leads to peripheral sensitization of joint sensory nerves and this heightened neural activity is the physiological basis of joint pain. The data also provide the first objective evidence that disease severity is a poor indicator of joint pain. Moreover, we have identified VIP6−28 and locally administered cannabinoids as potential treatments for OA pain. Future studies using these and other OA models are required to elucidate further the neurophysiological processes responsible for generating joint pain so that novel therapeutics may be developed allowing OA patients to lead a pain-free life.