Possible morphological substrates in the pathogenesis of arthritis

K. J. van Zwieten, I. Lambrichts, B. S. de Bakker, L. Kosten, S. De Munter, P. Gervois, K. P. Schmidt, P. Helder, P. L. Lippens

1 Department of Anatomy, Morphology, BioMed Institute, University of Hasselt, Diepenbeek, Belgium.
2 Department of Anatomy, Embryology and Physiology, Academic Medical Center, University of Amsterdam, The Netherlands.
3 HandShoe Mouse, Hippus NV, Rotterdam, The Netherlands.

Introduction, Material and Methods. To gain more insight into the morphogenesis of synovial joints, as well as into pathogeneses of joint diseases like Rheumatoid Arthritis (RA) and possibly also Osteoarthritis (OA), we surveyed human finger joint ontogeny from early developmental stages (1). High Resolution Magnetic Resonance Imaging (HR-MRI) of the adult proximal interphalangeal (PIP)-joint in an otherwise normal anatomical specimen of the finger was then analyzed. We also investigated the histology of a part of the PIP-joint capsule, the Proper Collateral Ligament (PCL).

Observational results. Before finger joint cavitation becomes obvious, interphalangeal interzones already indicate convexities at the future ‘ball’-side of each joint, and concavities at its future ‘socket’-side. By their broadness, these interzones also prefigure joint-capsule development. Later in ontogeny the increasing incongruences of the PIP-joint’s articular surfaces become more or less ‘compensated’ by meniscus-like vascularized synovial folds (2). According to present-day views, as formulated by Decker et al. (2014) in an authoritative review article, in these folds “Gdf5 expression would be activated along with other interzone-specific genes. Additional differentiation processes and mechanisms such as muscle movement would bring about cavitation and genesis of other joint tissues such as ligaments and other meniscus involving Gdf5- and Tgfbr2-positive and -negative cell progenies” (3). In adult interphalangeal joints, dorsal and palmar but also ulnar and radial synovial folds do persist, as HR-MRI slices reveal. During adult life, synovial membranes but also synovial folds (e.g., after micro-damage) may elicit chronic inflammatory processes that eventually lead to arthritis. This focused our attention on the normal vascularity of the articular capsules of finger joints.

PIP-arteriography and microscopy demonstrate microvascular articular networks. Capillaries accompanied by neurons, surrounded by connective tissues, pierce through the various collagenous fiber-bundles of the PIP-joint’s Proper Collateral Ligament. By Neurofilament Heavy Antibody staining, the accompanying neural structures were made visible in detail. Results, Conclusions. In PIP collateral ligaments, we found neurofilaments and oval-shaped lamellated corpuscles (ø ≈ 120 µ). Supposedly acting as mechanoreceptors, these may also produce cytokines and substance P, a neuropeptide involved in pain (4). As the PIP-joints of fingers in particular show symptoms of RA at early stages, these organs may therefore play a role in the pathogenesis of Rheumatoid Arthritis.

References


