



Rheumatoid arthritis revisited



ABSTRACT

The generally accepted theory of the etiopathology of rheumatoid arthritis has not led to a breakthrough so far, and the root cause of RA is still unknown. Surgical experience does not support the idea that the damage in the joint is caused by synovitis. Synovial tissue does not spread on the surface of the cartilage and thus destroy it, and neither is the erosion caused by the synovial tissue. Macroscopically and microscopically synovitis in RA is no different to synovitis in arthrosis. Perhaps the etiopathogenesis should once more be reconsidered.

For decades RA has been considered as an autoimmune reaction against the synovium. The fulminant inflamed synovium is thought to grow on the cartilage surfaces as a pannus destroying the chondroid tissue. However, peroperatively pannus is never seen on the cartilage surface nor does it erode the bone.

Moreover, in RA synovitis always resolves when the cartilage is gone or replaced with a total prosthesis. This occurs also in tenosynovitis when the tendon spontaneously ruptures.

Furthermore, rheumatoid and osteoarthritic synovitis cannot be differentiated neither macroscopically by a surgeon nor microscopically by a pathologist.

Then what causes the erosion? In the knee it is not in place of the most fulminant synovitis proximally but typically hiding under the meniscotibial ligament around tibial condyles.

When one lifts their leg to take a step with a swollen knee, the synovial fluid flows under the menisci, and when the heel is pressed to the ground, this space is tightly closed. The load of the body rapidly raises the pressure causing an extremely powerful flow around the tibial head. Could the erosion be a mechanical lesion caused by the flow?

In CMC2-3 there is no mobility and they are never affected, neither are patellar nor Achilles tendons – But only tendons which have a curved line of sliding like those in hands and behind malleoli.

Targets of manifestations of RA are constantly subject to great, versatile movement from joints to cardiac valves. The affected tissue might be the collagen. Is there something wrong in the collagen synthesis of those patients inheriting RA? Or is this a question of

microbial attack due to impaired inherited immune capacity? Or thinking of the tendons curving behind the malleoli, is it the linkage of the collagen fibres? And lastly, naturally, is RA an autoimmune disease at all?

Considering other autoimmune diseases, why do we have temporal but not radial arteritis? The temporal artery moves quite freely without any support from the surrounding tissue, so the arterial wall could be damaged due to millions of pulse waves. Similarly in the kidneys the pulse wave is strong and the mechanical load is immense in the long run causing glomerular nephritis. Analogically, post-infectious arthritis is a cartilage damage caused by bacteria or endotoxin, and here too synovitis is secondary. Ankylosing spondylitis is a stiffening lesion of the collagen tissue in spinal discs and pelvic joint capsules.

From a surgeon's point of view, in RA it is simply a question of collagen structure failing in its task: transiting and absorbing movement. The immunological event seen in so many structures previously thought to be autoimmune is secondary to the mechanical lesion. The immune reaction is not destructive, it aims to be reconstructive.

Conflict of interests statement

None.

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