THERAPY

Successful biologic therapy leads to an improvement in synovial tissue hypoxia

Treatment with tumor necrosis factor (TNF)-blocking agents can improve *in vivo* synovial hypoxia in patients with inflammatory arthritis who respond to this therapy. “These changes are strongly associated with vascularity and various measures of improvement in joint inflammation, which further strengthens the concept that hypoxia is an important event driving synovial inflammation,” says Douglas Veale, whose team published these findings in *Arthritis & Rheumatism*.

Inflamed synovial tissue is known to be profoundly hypoxic in individuals with inflammatory arthritis; in addition, the partial pressure of oxygen (pO_2) in their synovial tissue correlates inversely with markers of synovial inflammation.

The researchers recruited 20 patients with active inflammatory arthritis who were naive to biologic drugs. *In vivo* synovial tissue pO_2 values increased from baseline only in individuals who responded to anti-TNF treatment,

indicated by 28-joint disease activity score incorporating C-reactive protein levels (DAS28-CRP) of <2.6 at 3 months. The pO_2 synovial membrane values of nonresponders (DAS28-CRP >2.6) either decreased or remained the same. The increase in pO_2 observed in responders also correlated inversely with DAS28-CRP values, the extent of macroscopic synovitis, and numbers of CD4⁺ T cells and CD68⁺ macrophages.

The authors conclude that if hypoxia is an early event driving angiogenesis, or if it is a possible trigger factor in the pathogenesis of inflammatory arthritis, then perhaps very early intervention with TNF-blocking agents could restore the tissue to normal.

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Original article Kennedy, A. *et al.* TNF blocking therapy alters joint inflammation and hypoxia. *Arthritis Rheum.* doi:10.1002/art.30221