Surgery on the Rheumatoid Ankle Joint: Efficacy Versus Effectiveness

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KEYWORDS

• Rheumatoid arthritis • Synovectomy • Ankle arthrodesis
• Total ankle arthroplasty

Synovectomy, ankle arthrodesis (AA), and total ankle arthroplasty (TAA) are surgical options that address early and late stage diseases in the rheumatoid ankle joint. Synovectomy, for early stage disease, is considered prophylactic at times and is viewed with skepticism because of the chronic inflammatory process within joints that results in progressive tissue degradation and joint destruction. AA and TAA are the 2 most common procedures implemented for symptomatic end-stage joint destruction. The painful ankylosed ankle in patients with rheumatoid arthritis (RA) is one scenario in which the treatment of choice, ankle fusion for end-stage joint destruction, provides an outcome while considered definitive to that specific joint but is not preferred to the maintenance of motion. Total ankle replacement of early generation designs did not provide consistent desired results furthering AA as the treatment of choice. Newer generation ankle implant designs present an opportunity to reassess treatment algorithms for end-stage RA. The progressive disease process associated with RA complicates the long-term outcomes of all treatment options because it has systemic effects on bone, soft tissue, blood vessels, and viscera. The emergence of osteoimmunology provides a greater understanding of the RA disease process. Understanding the pathophysiology of RA gives the surgeon a greater insight as to how this disease can affect procedure outcome. Gait patterns in the patients with RA are significantly altered in end-stage disease as compared with healthy individuals (HI) and should be considered during procedure selection. The effect of RA on bone mineral density (BMD) affects the effectiveness of fixation, and an awareness of the biomechanical differences of various fixation techniques in bones of differing densities is crucial to obtaining stability. This article reviews synovectomy, AA, and associated concepts. A delineation of physiologic interactions associated with RA is provided. TAA is reviewed in the article by DiDomenico and Treadwell elsewhere in this issue.

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OSTEOIMMUNOLOGY

An understanding of the physiologic process of RA provides an insight as to how the disease process progresses and affects surgical outcomes. Interactions between the immune system and the skeletal system have led to the development of osteoimmunology. This field of study is particularly relevant to the understanding of RA, as an immune-mediated regulation of bone loss is a well-developed concept that allows for an understanding of the architectural changes in bones that occur within this disease process. Bone remodeling is regulated by molecular and cellular events that coordinate the dynamic balance between osteoblastic and osteoclastic activity.[1] Interference of this balance can result in bone loss as demonstrated in RA.[2] The combination of chronic immune activation and musculoskeletal tissue damage is present in RA.[3] Inflammation is a defining factor in RA especially in the most severe cases. Periarticular bone destruction, a central feature of RA, requires the presence of osteoclasts (OCs) in the joint because this is the only cell type that can remove calcium from bone.[1] OCs are present within the inflamed synovial tissue and the bone-pannus interface in RA.[4] RA is essentially a purely erosive disease with minimal indication of bone repair. The immune and skeletal systems have demonstrated various regulatory molecules in common, such as cytokines.[5] The physiology and pathology of one system affects the other. Because immune cells are formed in the bone marrow by interacting with bone cells, abnormal activation of the immune system can lead to synovial hyperplasia and bone destruction in RA.

The induction of OC precursors near the bone or joint surface to form OCs is mediated by factors in the local environment including within the synovium.[6] When these factors bind to OC precursors multiple genes are expressed that are vital to the function of OCs. As OCs undergo structural changes, they bind to the bone surface, acidify the microenvironment, decalcify the bone, mobilize the mineral content, break down the bone matrix, and then excrete the degradation products into the circulation. These products include solubilized calcium and phosphate, which are intricately involved in maintaining systemic homeostasis.

The expression of receptor activator of nuclear factor-κB ligand (RANKL), also known as osteoclast differentiation factor, osteoprotegerin (OPG) ligand, and tumor necrosis factor-related activation-induced cytokine, has been identified in the synovium of patients with RA, whereas it is not detected in the synovium of patients with other bone diseases.[7] RANKL is an essential stimulating signal for osteoclastogenesis and is involved in the activation of mature OCs.[8] RANKL in connection with macrophage colony-stimulating factor (M-CSF) aids in OC differentiation.

Cytokines are a category of signaling molecules that function extensively in cellular communication. Inflammatory cytokines, such as tumor necrosis factor (TNF)α, interleukin (IL)-1, and IL-6, can act to upregulate or accelerate bone destruction in RA.[9] TNF-α induces RANKL and also stimulates OC precursor cells to synergize with RANKL signaling while inhibiting osteoblasts to affect bone mass locally and systemically.[10] Erosion sites in RA demonstrate pooling of OC precursors in bone marrow cells adjacent to the invading pannus.[7] Synovial tissue in patients with RA is a source of RANKL further implying its involvement in bone erosion.

T-cell infiltration is a hallmark of RA synovium.[11] The bone-lymphocyte relationship has been well appreciated because the early development of lymphocytes occurs in bone. Typically there is no bone loss under normal T-cell response. T cells have an inhibitory effect on osteoclastogenesis via secretion of interferon (IFN)–γ and IL-4 that inhibit RANK and block osteoclastogenesis. However, in vivo studies have demonstrated that T-cell–derived RANKL is responsible for osteoclastogenesis and