Chapter 3: Basic Concepts and Therapeutics in (Nondrug) Musculoskeletal Care

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Pain, perhaps due to minor injury, recommendation from physicians and the pharmaceutical industry to use drugs as easy and heavily advertised drug solutions

Use of NSAIDs

Increased intestinal permeability: "leaky gut"


Immune complex formation and deposition

Immune activation and dysregulation (e.g., superantigens, NFkB, ERS, MitoDys)

Pain and inflammation: clinical presentation of "arthritis"

Clinically significant promotion of joint degeneration (Newman and Ling, Lancet 1985 Jul)

Inhibition of proteoglycan synthesis, induction of subchondral osteonecrosis

The vicious cycle of NSAID use: Pain prompts doctors and patients to use NSAIDS, which then promote joint destruction and increased intestinal permeability that promotes systemic inflammation which then contribute(s) to the perpetuation of joint pain.

- Promotion of hepatic and renal injury and failure: Chronic use of NSAIDs is an important risk factor for the development of renal failure. Hepatic injury is less common than NSAID-induced renal failure but can be achieved with higher drug doses (especially with the non-NSAID analgesic acetaminophen), coadministration of drugs, and concomitant consumption of alcohol.
- Death: NSAIDs are an impressively significant cause of death in America. According to the review by Singh, "Conservative calculations estimate that approximately 107,000 patients are hospitalized annually for nonsteroidal anti-inflammatory drug (NSAID)-related gastrointestinal (GI) complications and at least 16,500 NSAID-related deaths occur each year among arthritis patients alone. The figures for all NSAID users would be overwhelming, yet the scope of this problem is generally under-appreciated."

33 "At...concentrations comparable to those... in the synovial fluid of patients treated with the drug, several NSAIDs suppress proteoglycan synthesis... These NSAID-related effects on chondrocyte metabolism... are much more profound in osteoarthritic cartilage than in normal cartilage, due to enhanced uptake of NSAIDs by the osteoarthritic cartilage." Brandt KD. Effects of nonsteroidal anti-inflammatory drugs on chondrocyte metabolism in vitro and in vivo. Am J Med. 1987 Nov 20; 83(5A): 29-34
35 "This highly significant association between NSAID use and acetabular destruction gives cause for concern, not least because of the difficulty in achieving satisfactory hip replacements in patients with severely damaged acetabula." Newman et al. Acetabular bone destruction related to non-steroidal anti-inflammatory drugs. Lancet. 1985; 2: 11-4
37 "Histological observations suggest that cox-2 is required for normal endochondral ossification during fracture healing. Because mice lacking Cox2 form normal skeletons, our observations indicate that fetal bone development and fracture healing are different and that cox-2 function is specifically essential for fracture healing." Simon AM, Manigrasso MB, O’Connor JP. Cyclo-oxygenase 2 function is essential for bone fracture healing. J Bone Miner Res. 2002 Jun;17(6):963-76