

T R E A T M E N T O F

Rheumatoid Arthritis

Rheumatoid Arthritis (RA) is a chronic, debilitating disease that affects approximately 2.1 million Americans.³¹ Nearly 80 percent of all RA cases begin between the ages of 35-50 years and is 2-3 times more prevalent in women than men. Additionally, with RA there is a decrease in life expectancy of 5 to 15 years,³² secondary to development of comorbid illnesses, which may be exacerbated by the pathophysiologic mechanisms that underlie RA or by the medications used to treat the arthritis.

RA is a progressive systemic disease of unknown etiology most commonly characterized by a symmetrical inflammatory polyarthropathy with morning stiffness that may result in progressive destruction of articular and periarticular structures. At present, there is no cure for RA. Management goals are focused on preserving joint function and stopping disease progression. Current therapies are directed towards suppression of the inflammatory response. Persistent joint inflammation is believed to be associated with permanent articular cartilage and bone destruction, which can occur as early as 3 to 6 months after onset of RA. Although many patients improve symptomatically with conservative treatment during the early stages of disease, 10% or more are eventually severely disabled despite therapy. Non-steroidal anti-inflammatory drugs have long been used for symptomatic relief but

do not alter the long-term course of the disease. Low dose corticosteroids have a dramatic anti-inflammatory effect but do not prevent the progression of joint destruction, and long-term use is associated with significant side effects. During the past decade, disease-modifying antirheumatic drugs (DMARDs) such as hydroxychloroquine, methotrexate, sulfasalazine, leflunomide and cyclosporine have been used in early RA to halt progressive joint destruction. Methotrexate is generally considered the most effective DMARD of this group.

A significant advance for helping patients with RA has recently emerged from research on the cytokines tumor necrosis factor (TNF) and interleukin-1. These proinflammatory cytokines are overproduced in the joints of persons with RA and stimulate the production of synovial proteolytic enzymes in the joint that cause cartilage and bone

destruction. Four new biologic agents (etanercept, infliximab, anakinra, adalimumab) have been shown to be very effective inhibitors of these cytokines and thereby help prevent or slow progressive joint destruction, which leads to improved physical function in patients with RA. As seen in Table 4, the rates of inpatient admissions for RA has steadily decreased from 2000-2002, while rates for Osteoarthritis and Spine-Related arthritis continue to increase.

Furthermore, these new biological response-modifying agents act more rapidly in patients with early active RA.³³ This observation underscores the importance of early intervention in slowing or retarding joint damage, and confirms that early, aggressive treatment of active RA may be key to better long-term functional outcomes. Therefore, biological response-modifying agents show great promise for improving the course of RA.

Table 4
Rate of Change by Type of Arthritis/Treatment Inpatient Admissions 2000-2002

Type of Arthritis/Treatment	Total Admissions			Rate of Change
	2000	2001	2002	
All forms of Arthritis	30,671	34,223	35,990	15% increase
Osteoarthritis:	24,725	27,806	29,717	17% increase
• with Knee Replacement	15,312	17,422	18,882	19% increase
• with Hip Replacement	7,120	7,858	8,157	13% increase
Spine-Related Arthritis:	2,761	3,185	3,045	9% increase
Rheumatoid Arthritis:	899	885	855	5% decrease
• with Knee Replacement	192	166	158	18% decrease
• with Hip Replacement	114	96	80	29% decrease

Source: Pennsylvania Health Care Cost Containment Council



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- 13 Prepared by: Frank Ahern, Ph.D., Dept. of Biobehavioral Health, Penn State University, (814) 863-0185, f4@psu.edu.
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