Quick facts about RA treatment

There is currently no cure for RA[2]

Disease treatment goals centre on disease management[2]

Early diagnosis and rapid treatment are important in controlling disease progression and providing relief[2]

Most drugs for RA work by suppressing the immune system or masking pain[2]

➤ Fact sheet

Current treatment landscape

Although doctors are still unable to pinpoint the exact cause of RA, it is thought that genetic, environmental and hormonal factors all play a role.[1] There is no one way to diagnose RA, so the physician relies on a combination of medical history, physical examinations and laboratory tests.[2] Early diagnosis and treatment are important as joint damage begins to occur within the first two years of the disease.[3]

Treatment aims

Without a cure for RA, treatment goals centre on disease management and include:[2]

- Controlling disease progression
- Providing pain relief and reducing swelling
- Preventing joint damage and deformity
- Maintaining function of the affected joints and preventing disability

Treatment types

Most drugs for RA work by suppressing the immune system or masking pain.[2] Depending on the level of severity and stage of the disease, several different types of drug therapies may be used in monotherapy or combination:[2]

- Non-steroidal Anti-Inflammatory Drugs (NSAIDs)
- Corticosteroids
- Disease Modifying Antirheumatic Drugs (DMARDs)
- Anti-TNF therapies

Non-steroidal Anti-Inflammatory Drugs (NSAIDs)

NSAIDs reduce acute inflammation, decrease pain and improve function, and are available in both over the counter and prescription formulations.[3] NSAIDs inhibit the generation of prostaglandins by blocking cyclooxygenase enzymes.[3] Prostaglandins are mediators of inflammation and pain but also have an important role in maintenance of normal body functions.[3]

The NSAID class includes COX-2 inhibitors and selective COX-2 inhibitors exhibit safer gastrointestinal profiles than conventional non-selective NSAIDs, but these carry concerns of possible increases in cardiovascular risk.[3] It is important to note that NSAIDs alone do not change the course of the disease or prevent joint destruction.[3]

Corticosteroids

Corticosteroids have both anti-inflammatory and immunoregulatory activity, and are useful in the treatment of early RA as a temporary adjunctive therapy.[3] Frequent problems with corticosteroid therapy include; difficulty in discontinuing therapy, weight gain, increased blood pressure, increased blood sugar, or risk of cataracts, avascular necrosis of the bones, accelerated osteoporosis, and a cushingoid appearance.[3]

Recent studies suggest that low dose prednisone may have effects as a "disease modifying" agent in RA, especially when used in combination with other DMARD medications.[3]

Disease Modifying Antirheumatic Drugs (DMARDs)

DMARDs are the only agents shown to alter the course of RA and improve radiographic outcomes, although their effect may be more delayed in onset than NSAIDs or corticosteroids.[3] Methotrexate, considered the first-line DMARD agent, can be combined safely with nearly every other FDA approved DMARDs for RA.[3]

Methotrexate has a relatively rapid onset of action, good efficacy in reducing the signs and symptoms of RA, slowing or halting radiographic damage, a favourable toxicity profile, ease of administration, and relatively low cost. Some patients complain of gastrointestinal upset (nausea or diarrhoea), headache, fatigue, and feeling of being "wiped out" (also called methotrexate "fog").[3]

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Anti-TNF therapies

A number of anti-TNF therapies, also known as biological therapies, are currently available for use in patients with moderate to severe RA. They may be given alone, but are usually given in combination with methotrexate or another immunosuppressant. Anti-TNF therapies have proven to be effective treatments, with the potential to prevent further joint damage.[4] These therapies work by inhibiting the action of TNF-alpha, an inflammatory mediator, either directly or indirectly responsible for damaging the joint.[5] As with the other treatments, there are some side effects from the anti-TNF therapies including increased risk of infection and increased upper respiratory infection symptoms. Anti-TNF therapies are also not recommended for use in patients with demyelinating disease or with congestive heart failure.[3] Other undesirable side effects when treated with anti-TNF therapies may include fevers, chills, body aches, and headaches associated with the infusion of the antibody.[3]

For more information visit www.ucb.com

References

1. The Causes of Arthritis. The Arthritis Society. http://www.arthritis.ca/types%20of%20arthritis/ra/default.asp?s=1
2. Ruderman E. Rheumatoid Arthritis. American College of Rheumatology website, accessed May 2004. http://www.rheumatology.org/practice/clinical/patients/diseases_and_conditions/ra.asp
3. John Hopkins: Hopkins-arthritis.org: RA Treatment – Accessed March 2010 http://www.hopkins-arthritis.org/arthritis-info/rheumatoid-arthritis/rheum_treat.html
4. Moreland L. Treatment of Rheumatoid Arthritis with Recombinant Human Tumor Necrosis Factor receptor–fc Fusion Protein. NJEM 1997; 3:337:141-147
5. National Rheumatoid Arthritis Society (NRAS). Anti-TNF Treatment (Dr. Ernest Choy, arc Consultant Senior Lecturer in Rheumatology, GKT School of Medicine, King's College Hospital, London. http://www.rheumatoid.org.uk/article.php?article_id=55