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Review Article

An over view of rheumatoid arthritis

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ABSTRACT

Rheumatoid Arthritis (RA) is an autoimmune disease (the body's immune system attacks it's own tissue, including joints) over longer periods of time, can cause bone erosion and joint deformity. RA occurs in about 5 per 1000 people. At present any patients with clinical synovitis in at least one joint may have definite RA, requiring aggressive treatment if it is untreated or unaddressed it leads to complications such as permanent joint damage requiring arthroplasty, rheumatoid vasculitis, and syndrome requiring splenectomy. Here, we present brief summary of the treatment modalities to address the complications associated with Rheumatoid Arthritis.

Keywords: Rheumatoid Arthritis; Splenectomy; Arthroplasty

INTRODUCTION

Rheumatoid Arthritis (RA) is a chronic systemic autoimmune mostly seen in the females than the males, and frequently observed in the elderly. RA primarily affects the lining of the synovial joints and can cause progressive disability, premature death and socioeconomic burdens. Early diagnosis is considered as the key improvement index for most desirable outcomes such as reduced joint destruction, less radiologic progression no function disability, and disease modifying anti rheumatic drugs (DMARD) free remission. The treatment and outcome of RA influenced by the actions like the willingness of patients to seek medical advice, patient awareness of RA, the time for the patients from symptom onset to receiving appropriate treatment, the diagnostic capability of the physician to measure the disease activity. There are many composite scales such as the disease activity score using 28 joints (DAS – 28), Simplified Disease Activity Assessment Index (SDAI), and Clinical Disease Assessment Index (CDAI). Currently,

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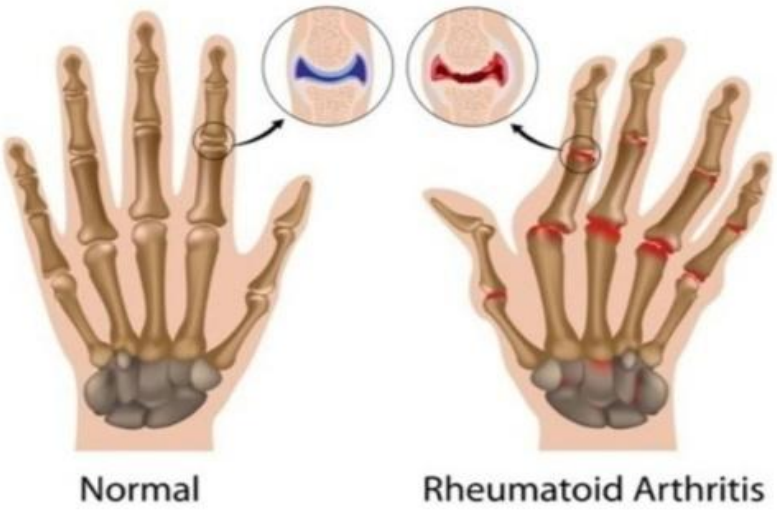
there is no treatment to cure the RA completely but earlier diagnosis and treatment of inflammatory arthritis is expected to prevent development of RA and even exert a curative effect for a proportion of patients on the other hand inappropriate treatment of patients who do not develop RA is harmful and should be avoided, so, it is important to predict the RA development in those Patients who have persistent arthritis. Over the last 20 years, the effectiveness of DMARDs has gained much attention as these can efficiently attenuate disease activity and subsequently decrease and/or delay joint deformity. Common symptoms of RA include morning stiffness of the affected joints for less than 30 minutes, fatigue, fever, and weight loss, joints that are tender, swollen and warm and rheumatoid nodules under the skin. The onset of this disease is usually from the age of 35 to 60 years with remission and exacerbation. Clinically, the diagnosis of RA can be differentiated from osteoarthritis (OA) as the affected areas in RA are the proximal interphalangeal (pip) and metacarpophalangeal (mp) joints; OA typically affects the distal interphalangeal (dip) joint. The goals of treatment for RA are to reduce joint inflammation and pain, maximize joint function and prevent joint destruction and deformity[1-3].



EPIDEMIOLOGY

In 2005, RA was prevalent in about 1.3 million adults in the United States, and 2 years later, it affected an estimated 1.5 million adults .More recent data on RA prevalence in the U.S. are not available yet in the literature. RA can occur in all races and ethnic groups. The prevalence of RA in developed countries is 0.5% to 1% of the population (0.6% in the U.S.).Women have a two-to- three times greater predisposition for developing RA compared with men. RA[4-7] onset generally occurs in middle age and is more common in older adults, but it can also develop in children and young adults. The lifetime risk of developing an inflammatory autoimmune rheumatic disease is 1 in 12 (8.3%) for women and 1 in 20 (5%) for men. Specifically, the lifetime risk of developing adult-onset RA is 1 in 28 (3.6%) for women and 1 in 59 (1.7%) for men .Over time, RA severity has declined, particularly owing to earlier diagnosis and more effective drug regimens, but trends in RA incidence, prevalence, and mortality vary based on the studied population.

ETIOLOGY

The exact cause of RA is still unknown, but genes, environmental factors, and hormones may be involved in its autoimmune development and progression. Certain risk factors appear to increase the risk of RA, including older age (highest incidence in people aged 60 years); gender (higher incidence in women); genetics (especially human leukocyte antigen [HLA] class II genotypes, such as HLA-DRB1); smoking (tobacco,

cigarettes); history of live births (higher RA risk with nulliparity); early life exposures (if mother smoked, child has greater risk of RA); and obesity (higher risk with increasing body weight). Patients who are seropositive for anticitrullinated protein antibodies (ACPAs) or rheumatoid factors (RFs) also have an increased risk of RA. Interestingly, women who breastfeed their children appear to have a lower risk of RA. Before the advent of effective disease-modifying antirheumatic drugs (DMARDs) and biological therapies, patients with RA had a higher likelihood of dying from premature atherosclerosis, cancer, and infection[8-10].

PATHOPHYSIOLOGY

Joint swelling in RA is usually synovial-membrane inflammation, with cytokine and chemokine involvement. The most relevant components in the inflamed space include tumor necrosis factor (TNF), interleukin-6 (IL-6), and granulocyte-macrophage colony-stimulating factor. Cytokines and chemokines induce or aggravate the inflammatory response by activating endothelial cells and promoting immune-system cell accumulation within the synovial compartment. Activated fibroblasts, B cells, T cells, monocytes, and macrophages can eventually trigger osteoclast generation via receptor activator of nuclear factor kappa-B ligand (RANKL), which is expressed on B cells, T cells, and fibroblasts. The RANK receptor is present on macrophages, dendritic cells, and preosteoclasts. In addition, the cartilage matrix within joints is eventually degraded by metalloproteinases and other enzymes[11-15].

DIAGNOSIS

In RA, signs and symptoms may overlap with other rheumatic diseases, but classification criteria can assist with diagnosis. Patients with RA will generally complain of multiarticular pain/aching, morning stiffness, tenderness/swelling, and bilateral/symmetrical joint involvement (e.g., both hands, both knees). Patients may also present with weight loss, fever, fatigue, and/or weakness. In addition to physical symptoms, the laboratory diagnosis (measurable sign) of RA has improved with the identification of highly specific biomarkers. Along with an elevated C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), the presence of autoantibodies (e.g., ACPA, RF) typically indicates more severe joint damage and increased mortality. Of note, RF is directly involved in mechanisms related to cytokine and macrophage activation. ACPAs form immune complexes that interact with RF, enhancing the effects of inflammation and subsequent joint destruction. While on treatment for RA, the concentrations of ACPA and RF should decrease. Patients will rarely become ACPA seronegative, but they can become RF seronegative.

These findings and laboratory values are incorporated in the classification criteria for RA, with joint involvement, serology, acute-phase reactants, and symptom duration as the main categories for consideration. An increasing number of affected joints (≥1 small joint), positivity of RF and/or ACPA, abnormal ESR and/or CRP, and duration of symptoms (≥6 weeks) are scored and calculated, with a score of ≥6 out of 10 being a definitive RA diagnosis.

Causes of RA

In a healthy person, the immune system fights invaders, such as bacteria and viruses. With an autoimmune disease like RA, the immune system mistakes the body's cells for foreign invaders and releases inflammatory chemicals that attack, in the case of RA, the synovium. That's the tissue lining around a joint that produces a fluid to help the joint move smoothly. The inflamed synovium gets thicker and makes the joint area feel painful and tender, look red and swollen and moving the joint may be difficult.

Researchers aren't sure why some people develop RA. They think that these individuals have certain genes that are activated by a trigger in the environment, like a virus or bacteria, or physical or emotional stress or some other external factor.

Symptoms

In the early stages, people with RA may not see redness or swelling in the joints, but they may experience tenderness and pain.

- These symptoms are clues to RA:
- Joint pain, tenderness, swelling or stiffness that lasts for six weeks or longer.
- Morning stiffness that lasts for 30 minutes or longer.
- More than one joint is affected.
- Small joints (wrists, certain joints in the hands and feet) are typically affected first.
- The same joints on both sides of the body are affected.
- Many people with RA get very tired (fatigue) and some may have a low-grade fever. RA symptoms may come and go. Having a lot of inflammation and other symptoms is called a flare. A flare can last for days or months.

RHEUMATOID ARTHRITIS TREATMENT

Rheumatoid Arthritis is a chronic disorder for which there is no known cure. Fortunately, in the last few years, a shift in strategy toward the earlier institution of disease modifying drugs and the availability of new classes of medications have

greatly improved the outcomes that can be expected by most patients. The goal of rheumatoid arthritis treatment now aims toward achieving the lowest possible level of arthritis disease activity and remission if possible, minimizing joint damage, and enhancing physical function and quality of life. The optimal treatment of RA requires a comprehensive program that combines medical, social, and emotional support for the patient. It is essential that the patient and the patient's family be educated about the nature and course of the disease. Treatment options include medications, reduction of joint stress, physical and occupational therapy, and surgical intervention.

Pharmacological Strategies

There are three general classes of drugs commonly used in the treatment of rheumatoid arthritis: non-steroidal anti-inflammatory agents (NSAIDs), corticosteroids, and disease modifying anti-rheumatic drugs (DMARDs). NSAIDs and corticosteroids have a short onset of action while DMARDs can take several weeks or months to demonstrate a clinical effect. DMARDs include methotrexate, sulfasalazine, leflunomide (Arava), etanercept (Enbrel), infliximab (Remicade), adalimumab (Humira), certolizumab pegol (Cimzia), golimumab (Simponi), abatacept (Orenci), rituximab (Rituxan), tocilizumab (Actemra), anakinra (Kineret), antimalarials (e.g. Plaquenil). Other immunomodulators are occasionally used including azathioprine (Imuran) and cyclosporine. Because cartilage damage and bony erosions frequently occur within the first two years of disease, rheumatologists now move aggressively to a DMARD agent early in the course of disease, usually as soon as a diagnosis is confirmed.Analgesic drugs are also sometimes helpful in decreasing pain until DMARDs effect.

Non-steroidal Anti-inflammatory Agents (NSAIDs)

The major effect of these agents is to reduce acute inflammation thereby decreasing pain and improving function. All of these drugs also have mild to moderate analgesic properties independent of their anti-inflammatory effect. It is important to note however that these drugs alone do not change the course of the disease of rheumatoid arthritis or prevent joint destruction.

Aspirin is the oldest drug of the non-steroidal class, but because of its high rate of GI toxicity, a narrow window between toxic and anti-inflammatory serum levels, and the inconvenience of multiple daily doses, aspirin's use as the initial choice of drug therapy has largely been replaced by other NSAIDs. There are a large number of NSAIDs from which to choose, and at full dosages all are potentially equally effective. Likewise, the toxicities of the currently available NSAIDs are similar. However, there is a great deal of variation in tolerance and response to a particular NSAID. Many different NSAIDS are available, some over the counter including ibuprofen (Advil , Motrin, Nuprin) and naproxen (Alleve) and many others are available by prescription including meloxicam (Mobic), etodolac (Lodine), nabumetone (Relafen), sulindac (Clinoril), tolementin (Tolectin), choline magnesium salicylate (Trilasate), diclofenac (Cataflam, Voltaren, Arthrotec), diflusinal (Dolobid), indomethacin (Indocin), ketoprofen (Orudis, Oruvail), meloxicam (Mobic), oxaprozin (Daypro), and piroxicam (Feldene). Longer acting NSAIDs that allow daily or twice daily dosing may improve compliance. The NSAID class also includes drugs known as COX-2 inhibitors that are also effective in controlling inflammation. Only one of these agents is currently available in the United States (celecoxib, Celebrex) while additional compounds are available in other countries (etoricoxib, Arcoxia; lumiracoxib, Prexige). These drugs were designed to decrease the gastrointestinal risk of NSAIDS, but concerns of possible increases in cardiovascular risk with these agents has led to the withdrawal of two of these drugs from the market (rofecoxib, Vioxx; valdecoxib, Bextra).

Mechanism: NSAIDs inhibit the generation of prostaglandins by blocking cyclooxygenase enzymes, COX-1 and COX-2. Prostaglandins are mediators of inflammation and pain but also have important roles in maintenance of normal body functions including protection from stomach acid, maintenance of kidney blood flow, and contributing to platelet stickiness and vascular function. COX-2 selective inhibitors selectively block prostaglandins generated via COX-2 which have prominent roles in inflammation.

Dosage: While in some cases, lower doses of NSAIDS are effective, in rheumatoid arthritis and other forms of inflammatory arthritis a higher dose is often required to decrease inflammation. A lower dosage can initially be used if inflammation is mild, if mechanical pain is the major problem, if the patient is elderly or if the patient suffers from conditions that increase the risk for toxicity (see below). If a particular preparation is ineffective after a 4-week trial or is not tolerated, then another NSAID can be initiated. No one NSAID has been demonstrated to be better than another for the treatment of rheumatoid arthritis nor have the COX-2 agents been shown to be superior to traditional NSAIDS in terms of effectiveness.

Usual Time to Effect: Although these agents have anti-inflammatory effect within hours, a reasonable trial period is a few weeks to 1 month.

Side Effects: The most common toxicity of NSAIDs is gastrointestinal disturbance which may clinically include burning, belching, or irritation, but which can represent irritation of the lining of the stomach, erosions, and even ulcerations that can result in bleeding. While taking the medication with food may eliminate some of these symptoms, this does not decrease a risk of bleeding. The co-administration of medications known as proton pump inhibitors such as omeprazole (Prilosec), Lansoprazole (PPrevaci), Esomeprazole (Nexium), Pantoprazole (Protonix), and Rabeprazole (Aciphex), and a medication that provides back protective prostaglandins called misoprostol (Cytotec) can also decrease gastrointestinal bleeding associated with these medications. Misoprostol is combined in a single pill with the NSAID diclofenac (AArthrote). Selective COX-2 inhibitors exhibit safer GI profiles than conventional non-selective NSAIDs.

Because prostaglandins play a role in the regulation of the blood flow in the kidneys and maintenance of glomerular filtration, NSAIDs can also impair renal function in certain patients leading to salt retention, edema, and increased blood pressure. The patients at highest risk are those with fluid imbalances or with compromised kidney function (e.g., heart failure, diuretic use, cirrhosis, dehydration, and renal insufficiency). NSAIDs may also increase cardiovascular risks by their effects on blood pressure and additional effects on vascular beds. Thus, the use of this class of medications must into account their relative risks in an individual patient of gastrointestinal damage versus potential cardiovascular risk factors.

Corticosteroids

Corticosteroids (such as prednisone; methylprenisolone, Medrol) have both anti-inflammatory and immunoregulatory activity. They can be given orally, intravenously, intramuscularly or can be injected directly into the joint. Corticosteroids are useful in early disease as temporary adjunctive therapy while waiting for DMARDs to exert their anti-inflammatory effects. Corticosteroids are also useful as chronic adjunctive therapy in patients with severe disease that is not well controlled on NSAIDs and DMARDs. The usual dose of prednisone is 5 to 10mg daily. Although prednisone can be started at higher doses (15 to 20mg daily), attempts should be made to taper the dose over a few weeks to less than 10mg daily. Once started, corticosteroid therapy may be difficult to discontinue and even at low doses. Some patients are very sensitive to the tapering of prednisone which may be done slowly over a few weeks. Weight gain and a cushingoid appearance (increased fat deposition around the face, redness of the cheeks, development of a "buffalo hump" over the neck) is a frequent problem and source of patient complaints. Other side effects of prednisone include weight gain, increased blood pressure, increased blood sugar, increased risk of cataracts, and avascular necrosis of bones.

Steroid medications are also associated with accelerated osteoporosis even with relatively low dose prednisone at doses of 10 mg daily. Patients with and without osteoporosis risk factors on low dose prednisone should undergo bone densitometry (DEXA Scan) to assess fracture risk. Bisphosphonates such as alendronate (Fosamax), risedronate (Actonel), ibandronate (Boniva) are recommended to prevent and/or treat osteoporosis in addition to adequate calcium and vitamin D supplementation.

Higher doses of prednisone are rarely necessary unless there is a life-threatening complication of RA and, if used for prolonged periods, may lead to serious steroid toxicity. Although a few patients can tolerate every other day dosing of corticosteroids which may reduce side effects, most require corticosteroids daily to avoid symptoms. Once a day dosing of prednisone is associated with fewer side effects than the equivalent dose given twice or three times daily. Generally, steroids are given in the morning upon wakening to mimic the body's own steroid surge. Repetitive short courses of high-dose corticosteroids, intermittent intramuscular injections, adrenocorticotropic hormone injections, and the use of corticosteroids as the sole therapeutic agent are all to be avoided.

Intra-articular corticosteroids (e.g., triamcinolone or methylprednisolone and others) are effective for controlling a local flare in a joint without changing the overall drug regimen.

Disease Modifying Anti-rheumatic Drugs (DMARDS)

Although both NSAIDs and DMARD agents improve symptoms of active rheumatoid arthritis, only DMARD agents have been shown to alter the disease course and improve radiographic outcomes. DMARDs have an effect upon rheumatoid arthritis that is different and may be slower. In most cases, when the diagnosis of rheumatoid arthritis is confirmed, DMARD agents should be started. The presence of erosions or joint space narrowing on x-rays of the involved joints is a clear indication for DMARD therapy, however one should not wait for x-ray changes to occur.

The currently available drugs includes,

Methotrexate

Methotrexate is now considered the first-line DMARD agent for most patients with RA. It has a

relatively rapid onset of action at therapeutic doses (6-8 weeks), good efficacy, favorable toxicity profile, ease of administration, and relatively low cost. When looking at groups of patients on different DMARDS, the majority of patients continue to take Methotrexate after 5 years, far more than other therapies reflecting both its efficacy and tolerability. Methotrexate is effective in reducing the signs and symptoms of RA, as well as slowing or halting radiographic damage. It was as effective as leflunomide and sulfasalazine in one study, and its effectiveness given early and in higher doses approached the efficacy of etanercept and adalimumab as single therapies in terms of signs and symptom improvement. Methotrexate is also effective in many other forms of inflammatory arthritis including psoriatic arthritis and other spondyloarthopathies, and is used in many other autoimmune diseases.

Mechanism: The anti-inflammatory effects of methotrexate in rheumatoid arthritis appear to be related at least in part to interruption of adenosine and possible effects on other inflammatory and immunoregulatory pathways. The immunosuppressive and toxic effects of methotrexate are due to the inhibition of an enzyme involved in the metabolism of folic acid, dihydrofolate reductase.

Dosage: Dosing typically begins at 12.5-15 mg once per week. A dose escalation to 20 mg within the first three months is now fairly well accepted in clinical practice. Maximal dose is usually 25 mg per week but is sometimes increased further to 30 mg. Methotrexate can be given orally or by subcutaneous injection. The latter route of administration can be advantageous for patients who have methotrexate-associated nausea. Patients starting methotrexate should be carefully evaluated for renal insufficiency, acute or chronic liver disease, significant alcohol intake or alcohol abuse, leukopenia (low white blood cell counts), thrombocytopenia (low platelet counts), or untreated folate deficiency.

*Usual Time to Effect:*The onset of action is seen in as early as 4 to 6 weeks. However the dose required to achieve a response is variable in individual patients and may require 4-6 weeks after a dose increase to determine if the drug is working. A trial of 3 to 6 months at an increased dose (e.g. 20 mg/wk) is suggested. In patients with partial responses to methotrexate, additional medications are usually added to rather than substituted for methotrexate to achieve combination therapies.

Side Effects: Fortunately, the most serious complications of methotrexate therapy: hepatic cirrhosis, interstitial pneumonitis, and severe myelosuppression are quite rare, especially with proper monitoring. Stomatitis and oral ulcers, mild alopecia and hair thinning, and GI upset may occur and are related to folic acid antagonism. These side effects can be improved with folic acid supplementation. Folic acid given at a dose of 1mg daily does not diminish the efficacy of methotrexate and is routinely given with methotrexate to decrease these side effects. Some patients complain of headache, fatigue, and feeling "wiped out" (also called methotrexate "fog"). These side effects can often be overcome by increasing folic acid or using an activated form of folic acid known as folinic acid (leukovorin) given as a 5mg dose 12 hours and sometimes 24 hours after methotrexate is given. Some patients complain of GI upset (nausea or diarrhea) with oral methotrexate. This may be lessened when methotrexate is taken at night. In most cases this is completely eliminated when methotrexate is given by subcutaneous administration.

Before starting methotrexate, baseline studies should include complete blood count, liver chemistries, serum creatinine, hepatitis B and C serologies, and chest X-ray. Routine toxicity monitoring should include a CBC, liver profile, serum albumin and serum creatinine every 4-8 weeks.

Methotrexate can be combined safely with nearly every other FDA-approved DMARDs for RA, including sulfasalazine, hydroxychloroquine, TNF inhibitors, abatacept, rituximab, tocilizumab, anakinra, and leflunomide. In all clinical trials combining methotrexate with one of these DMARDs, no unexpected toxicities or synergistic toxicities were observed with the exception of higher liver toxicity with leflunomide which is also metabolized by the liver.

Hydroxychloroquine

Hydroxychloroquine is an antimalarial drug which is relatively safe and well-tolerated agent for the treatment of rheumatoid arthritis. Chloroquine is another antimalarial agent that is also sometimes used. Because these drugs have limited ability to prevent joint damage on their own, their use should probably be limited to patients with very mild, seronegative, and nonerosive disease. Hydroxychloroquine is sometimes combined with methotrexate for additive benefits for signs and symptoms or as part of a regimen of "triple therapy" with methotrexate and sulfasalazine.

Mechanism: The mechanism of action of antimalarials in the treatment of patients with rheumatoid arthritis is unknown but is thought to involve changes in antigen presentation or effects on the innate immunesystem.

Dosage: Hydroxychloroquine (Plaquenil) is the drug of choice among antimalarials. Chloroquine is not commonly used because of greater toxicity on the eye. The usual dose of Plaquenil is 400mg/day but 600mg/day is sometimes used as part of an induction regimen. It may be prescribed as a single daily dose or in divided doses twice per day.

Usual Time to Effect: A period of 2 to 4 months is usual. Most agree that if a patient shows no response after 5-6 months that this should be considered a drug failure.

Side Effects: The most important toxicities are on the eyes: corneal deposits, extraocular muscular weakness, loss of accommodation (and sensitivity to light), and a retinopathy that may progress to irreversible visual loss. Ocular toxicity is exceedingly rare, occurring in only 1 out of 40,000 patients treated at the doses recommended. Patients with underlying retinopathies or risks may not be good candidates for antimalarial drugs. Baseline ophthalmologic examination and a follow-up examination every 12 months are recommended during the period of treatment.

Sulfasalazine

Sulfasalazine **(Azulfidine)** is an effective DMARD for the treatment of RA. Its effectiveness overall is somewhat less than that methotrexate, but it has been shown to reduce signs and symptoms and slow radiographic damage. It is also given in conjunction with methotrexate and hydroxychloroquine as part of a regimen of "triple therapy" which has been shown to provide benefits to patients who have had inadequate responses to methotrexate alone. Sulfasalazine is also used in the treatment of inflammatory bowel disease and spondyloarthropathies. Its mechanism of action in RA is unknown. Some of its effects may be due to folate depletion.

Dosage: The usual dose is 2-3 grams per day in a twice daily dosing regimen. The dose may be initiated at 1 gram per day and increased as tolerated.

Usual Time to Effect: It may take 6 weeks to 3 months to see the effects of sulfasalazine.

Side effects: Sulfasalazine may cause hypersensitivity and allergic reactions in patients who have experienced reactions to sulfa medications. Mild gastrointestinal complaints are commonly seen and these can be decreased by using enteric coated formulations or administration of the medication with meals. Occasionally, mild cytopenias are seen. Patients may be screened before the use of sulfasalazine for a deficiency of the enzyme glucose-6-phosphate dehydrogenase (G6PD) which may predispose patients to red blood cell hemolysis and anemia. Blood monitoring is typically every 1-3 months depending on dose. Though sulfasalazine may cause increases in liver function tests, it is generally considered a preferable agent to methotrexate in patients with liver disease or in patients who have hepatitis B or C.

New medications: The newest drugs for the treatment of rheumatoid arthritis are the Janus Kinase (JAK) Inhibitors, which are FDA approved under the brand names Rinvoq, Olumiant, and Xeljanz.

Surgery

If medications fail to prevent or slow joint damage, you and your doctor may consider surgery to repair damaged joints. Surgery may help restore your ability to use your joint. It can also reduce pain and improve function.

Rheumatoid arthritis surgery may involve one or more of the following procedures:

Synovectomy: Surgery to remove the inflamed lining of the joint (synovium) can be performed on knees, elbows, wrists, fingers and hips.

Tendon repair: Inflammation and joint damage may cause tendons around your joint to loosen or rupture. Your surgeon may be able to repair the tendons around your joint.

Joint fusion: Surgically fusing a joint may be recommended to stabilize or realign a joint and for pain relief when a joint replacement isn't an option.

Total joint replacement: During joint replacement surgery, your surgeon removes the damaged parts of your joint and inserts a prosthesis made of metal and plastic.

Surgery carries a risk of bleeding, infection and pain.

Lifestyle and home remedies

You can take steps to care for your body if you have rheumatoid arthritis. These self-care measures, when used along with your rheumatoid arthritis medications, can help you manage your signs and symptoms:

Exercise regularly. Gentle exercise can help strengthen the muscles around your joints, and it can help fight fatigue you might feel. Check with your doctor before you start exercising. If you're just getting started, begin by taking a walk. Avoid exercising tender, injured or severely inflamed joints.

Apply heat or cold. Heat can help ease your pain and relax tense, painful muscles. Cold may dull the sensation of pain. Cold also has a numbing effect and can reduce swelling.

Relax. Find ways to cope with pain by reducing stress in your life. Techniques such as guided imagery, deep breathing and muscle relaxation can all be used to control pressure

ALTERNATIVE MEDICINE

Some common complementary and alternative treatments that have shown promise for rheumatoid arthritis include:

FISH OIL - Some preliminary studies have found that fish oil supplements may reduce rheumatoid arthritis pain and stiffness. Side effects can include nausea, belching and a fishy taste in the mouth. Fish oil can interfere with medications, so check with your doctor first.

PLANT OILS - The seeds of evening primrose, borage and black currant contain a type of fatty acid that may help with rheumatoid arthritis pain and morning stiffness. Side effects may include headache, diarrhea and gas. Some plant oils can cause liver damage or interfere with medications, so check with your doctor .

TAI CHI - This movement therapy involves gentle exercises and stretches combined with deep breathing. Many people use tai chi to relieve stress in their lives. Small studies have found that tai chi may improve mood and quality of life in people with rheumatoid arthritis. When led by a knowledgeable instructor, tai chi is safe. But don't do any moves that cause pain.

CONCLUSION

Early diagnosis and intervention are essential for the prevention of serious damage and loss of essential bodily functions. The treating physician should consider adhering to treat-to-target (T2T) recommendations, by first outlining the aims and then implementing the protocols to achieve and assess them. Furthermore, early referral to a specialist can help to ensure better treatment outcomes. With advances in the field of molecular medicine, we have a better understanding of disease mechanisms which can aid in the designing of more effective treatments. Old treatment modalities have been optimized and new ones have been produced. Gene array analysis is proving beneficial in finding out which patients will be more responsive to specific medications. This customization will allow for more rapid treatment as well as decrease the likelihood of disease progression during the experimental phase to seek an appropriate treatment for a particular patient. Gene array analysis is also being used to determine which patients are at greater risk for more aggressive forms of RA. It is foreseen that treatment methods will face tremendous improvements in the management of RA.

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