Review Article

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Reactive Arthritis in Chidren

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Abstract

The term reactive arthritis was first introduced in 1969 as to describe a sequel to an old infection, usually being genitourinary or GI infection. This condition has a diverse clinical manifestations. Initially it was know as Reiter's disease or Fiessinger Leroy disease. It is a member of spondyloarthritis family. HLA-B27 is a known predisposing genetic factor and is triggered by systemic infection. CREG-B7 group of antigens are found in B27 negative patients and these antigens are cross reactive to B27. Long term follow up is needed in children to determine if more number of children develop recurrent arthritis or any other disease like psoriatic arthritis, ankylosing spondylitis etc. cytokines like TNF alpha and interferon secrete T helper cells which help in eliminating the bacteria. NSAIDs, corticosteroids and antibiotics play a role in mainstay treatment. Reactive arthritis is characterized by both articular and extra-articular symptoms. Extra-articular manifestations include genitourinary symptoms, ocular symptoms, skin manifestations and cardiac manifestations. The diagnosis of this conditions is mostly clinical. Eliminating the infectious agent is the mainstay of the treatment followed by NSAIDs, corticosteroids and DMARDs in severe cases not responding to primary treatment is important.

Keywords: Reactive arthritis; Spondyloarthritis; NSAID; Children.

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INTRODUCTION

Reactive arthritis and post-infectious arthritis are defined as swelling of joints caused by a sterile inflammatory reaction following a recent or remote infection.[1-5] This condition usually occurs after an enteric infection with Salmonella species, Shigellaflexneri, Yersinia enterocolitica, Campylobacter jejuni, or GU tract infection with Chlamydia trachomatis. Majority of the patients with reactive arthritis are positive for HLA-B27

antigen. The cause of reactive arthritis is found to be due to Incomplete elimination of bacteria and its products, such as DNA. Ramesh BN et al[6] stated that rheumatoid arthritis (RA) is an autoimmune disease and a symmetrical polyarticular disease of unknown cause that affects mainly the diarthrodial joints, which is characterized by chronic swelling of the synovial joints. Colmegna I et al[5] stated that In 4th century B.C. Hippocrates was probably the first person who mentioned the term re active arthritis (ReA) when he noted that "Young men do not suffer from gout until they initiated sexual activity". Reactive arthritis is a joint inflammation developing after or during an infection elsewhere in the body, but the organism cannot be isolated from the joint. Reactive arthritis was observed to be inlink with gastrointestinal infections with bacteria such as Shigella, Salmonella, and Campylobacter species as well aswith genitourinary infections especially with Chlamydia trachomatis. Ajene AN et al[7] did systematic review in 2013 in which it was found that the mean incidence of reactive arthritis was 12, 12 and 9 cases per 1,000 of Salmonella, Shigella and Campylobacter infections respectively. This condition usually develops 14-28 days after a GI or genitourinary infection. Inman R. D et al[8] said that ReA is a pathological process in which there is an interaction between environmental and genetic factors. Inflammation occurs in joints, axial skeleton, skin, mucous membranes, gastrointestinal tract, and eyes. There is 50 times increased chance of developing reactive arthritis in patients with HLA-B27 positive. Sieper J et al[12] said that The arthritogenic peptide hypothesis states that there are microbial antigens which mimic certain self-antigens in the host, causing HLA-B27-specific CD8 T lymphocytes to react and leading to swelling of the joints. This would be due to molecular mimicry, a conditionin which there is a structural similarity between foreign and self-antigens that cause foreign peptides to give rise to a B or T-cell auto reaction.[13] Bas et al.[14] suggested that the cytokine reaction in HLA-B27 positive patients may have a part in improper elimination of the pathogen, which may lead to more severe or chronic arthritis.

CLINICAL FEATURE

The onset of this condition is usually sudden and characterized by lethargy, malaise and fever. The major presenting symptom of reactive arthritis is oligoarthritis mainly involving the lower extremities and is assymetrical. The inflammation causes pain, swelling, stiffness and redness of the joints that are involved. Non-joint areas involved in reactive arthritis are eyes, genitals, urinary tract, skin, large bowel, and the aorta. Conjunctivits and uveitis presents with redness of the eyes, pain, irritation and blurred vision. Urinary tract inflammation commonly involves the urethra. Minority of patients with reactive arthritis, especially those with chronic arthritis, will eventually develop cardiac manifestations including pericarditis and aortic regurgitation. Reiter triad includes urethritis, conjunctivitis, and arthritis may occur. Leirisalo-Repo M et al[9] described that Sacroiliitis occurs in approximately one third of the patients with urogenital reactive arthritis and in one tenth of patients with enteric reactive arthritis. Mucocutaneous lesions are observed in more than 50% of patients. Kim PS et al[10] described that Keratodermablennorhagica is highly specific of reactive arthritis. These are thepustular lesions, which are classically found on plantar regions, which may turn scaly and hyperkeratotic and coalesce to form psoriatic plaques. Circinatebalanitis is distinguished by painless, shallow psoriasiform lesions over the glans or shaft of the penis.

INVESTIGATIONS

Reactive arthritis cannot be diagnosed by a single lab test. ESR and CRP are elevated at the onset of the disease and later may become normal in the chronic stage of thedisease. There may be mild leukocytosis and anaemia in the early phase. The rheumatoid factor is usually negative. Gram staining and bacterial culture of the synovial fluid should be performed to differentiate from septic arthritis.[11-14]

TREATMENT

Identifying and eliminating the underlying infectious agent using appropriate antibiotics should be the main goal of the treatment. NSAIDs, Corticosteroids and immunosuppressants are needed for patients with severe reactive symptoms who do not respond to initial treatment. Sulfasalazine is beneficial in some patients. Methotrexate can be given in patients who present with rheumatoid likearthritis. Meyer et al.[15] has done a study in 10 patients with refractory Re A who did not respond to traditional drug therapy. All 10 patients were treated with anti-TNF antibodies, including five with infliximab, four with etanercept and one with adalimumab. In nine cases, treatment was effective, with significantly decreasing CRPand reduced joint symptoms. One

patient did not respond to infliximab. Sieper J et al[16] stated that in case of ReA patients with tendinitis or finger or toe inflammation, with no improvement in condition with NSAIDs and anti-rheumatic drugs, then the anti-TNF antibody should be taken into consideration.

CONCLUSION

Reactive arthritis is characterized by both articular and extra-articular symptoms. Extra-articular manifestations include genitourinary symptoms, ocular symptoms, skin manifestations and cardiac manifestations. The diagnosis of this conditions is mostly clinical. Eliminating the infectious agent is the mainstay of the treatment followed by NSAIDs, corticosteroids and DMARDs in severe cases not responding to primary treatment is important.

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