Reactive arthritis: causes, features and treatments

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Reactive arthritis

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Reactive Arthritis

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Introduction

Reactive arthritis (ReA) is defined as an episode of peripheral arthritis of more than one 1- month duration occurring in association with conjunctivitis and urethritis and/or cervicitis. It is triggered by an infection , most often in the gastrointestinal or urogenital tract. It is also known as Reiter’s syndrome, Feissinger – – Leroy’s disease, Brodie’s syndrome and conjunctivo-urethro-synovial syndrome. The term ReA Reactive arthritis was originally introduced to define a sterile joint inflammation during and after an infection elsewhere in the body. The definition was later modified since nucleic acids and bacterial antigens were found in the inflamed joints. [2]

Etiology Ae tiology

Reactive arthritis (ReA) follows an infection in the urogenital tract (venereal form) or gastrointestinal tract (dysenteric form). The venereal form follows recent sexual contact, whereas the dysentric dysenteric form is associated with a wide variety of intestinal pathogens and non-specific diarrh o eal illnesses. The most common organisms implicated are as follows:

1. Post Post- dysenteric form: Salmonella (different serotypes), *Yersinia tuberculosis , Shigella flexneri , Shigella S . sonnei ,* and *Campylobacter jejuni.* These organisms are found to be HLA HLA – B – 27 – dependent. Hence, Individuals individuals with HLA-B27 positivity are strongly predisposed to develop the disease .
2. Post Post- venereal form: *Chlamydia trachomatis* .

Some newer organisms have been implicated recently in causation of reactive arthritis ReA , namely *Chlamydia C . pneumonia , Mycoplasma hominis , Mycoplasma M . fermentans , N e isseria Gonorrhoea g onorrhoea e , Borrelia burgdorferi , Clostridium difficile , β-h a emolytic streptococci, Propionibacte r ium acnes , Escherischia Escherichia coli , Helicobacter pylori , Calmette Calmette – Guerin bacillus , Brucella abortus , Leptospira , Bartonella , Troph e y r e y ma whippeli , Gardnerella vaginalis , Giardia lamblia.* [3]

Drugs are generally not implicated in the a etiology of reactive arthritis ReA ; , however , a single case of Lithium lithium precipitating pre-existing Re A 1 : Kindly check for clarity> a OK cti ve arthritis has been described. â ´

Pathobiology

The prevalence of Re A active arthritis is estimated to be 0. 1% worldwide.

The disease mainly affects people in the 2 nd – 4 th second to fourth decade of life. The Infection i nfection occurs 1 – – 4 weeks following genitourinary infection , with a male – – female ratio of 9: 1. The Enteric e nteric type has an equal incidence in both males and females. â ¶

Systemic Features f eatures

The disease primarily affects the joints, eyes â , the skin and genitalia. Rarely, patients present with cardiac, renal , and neural abnormalities.

Arthritis

Articular manifestations are most commonly of an acute, non-destructive oligoarthritis usually affecting the large joints of the lower limbs which persists for 4 – – 5 months. ‘ Sausage digit’ or diffuse swelling of an entire toe/finger occurs in 16% of patients. Enthesitis is another characteristic feature of patients with ReA. It is defined as an inflammation of the ligaments and tendons at their site of insertion into the bone. Patients may also develop heel pain and achilles A chilles tendonitis. Sacroili i tis is another distinctive feature of the disease which results in a low back pain. [8-10]

Urethritis

Re A active arthritis usually follows 1 – – 3 weeks after an episode of urethritis. Urethritis may occur even in post post – dysenteric cases. The non non – specific urethritis presents with mild non-purulent urethral discharge. H a emorrhagic cystitis and prostatitis may develop in a few patients. In females, it manifests as cervicitis associated with cervical discharge. Rarely, bleeding and abdominal pain may occur. â µ

Mucocutaneous lesions

Keratoderma blen *n* orrhagica or Pustulosis p ustulosis palmoplantaris is a specific cutaneous lesion in ReA. Patients present with pustules over the

palms and soles which are gradually covered with thick horny crusts. Lesions may coalesce. Psoriasiform lesions are also common (Fig. 58. 1) . The biopsy of of skin lesions with acanthosis and epidermal neutrophilia (Fig. 58. 2) Circinate balanitis is a painless geographic dermatitis occurring over the glans penis (Fig. 58. 3 1). In addition, small , shallow ulcers are seen over the glans and urethral meatus and also over the oral cavity. Nail changes are a common finding and include subungual hyperkeratosis, onycholysis, ridging and nail shedding. [10, 11]

Visceral lesions

Visceral involvements mainly include the cardiac, renal and neural system s . Cardiovascular manifestations present as conduction delays and aortic disease. Proteinuria, microh a ematuria, aseptic pyuria, and rarely, glomerulonephritis occur when the renal system is involved. Transient neurologic dysfunction such as cranial or peripheral nerve palsies have been described in some patients. [10]

The disease is usually self self – limiting. The joint manifestations regress completely within a few months (3 – – 5 months). Enthesopathy, balanitis and psoriatic lesions may persist even after joint inflammation has subsided. Recurrences are common. Some patients develop chronic polyarthritis, usually HLA HLA – B – 27 – positive individuals. [12]

Ocular Features f eatures

Bilateral mucopurulent conjunctivitis is the most common ocular manifestation of ReA that occurs in more than 50% of patients. It is one of

the important components of the triad of the disease. Occasionally, the conjunctivitis may be purulent but remains transient, mild and associated with a sterile discharge. It subsides within 1 – – 4 weeks. Acute anterior uveitits uveitis may be found in about one-fifth of cases , especially in those who are positive for HLA-B27. [7] Other ocular complications of ReA include keratitis, corneal ulcer with or without hypopyon, episcleritis, scleritis, papill o edema, retinal o edema, retinal vasculitis and retrobulbar neuritis. [13]

Vision is usually impaired from corneal scar or recurrent chronic uveitis causing secondary glaucoma, complicated cataract or cystoid s macular o edema. [1] â ´

Diagnosis

Laboratory findings in ReA are non-specific and do not usually provide a conclusive diagnosis regarding the a etiology.

Prognosis

Individuals who are HLA HLA – B27 – positive have a more severe disease form . Male gender and a positive family history for spondyloarthropathies, ankylosing spondylitis and recurrent episodes of arthritis are indicators of a bad prognosis. [9]

Treatment

Patient education has plays a major role in patients with Re A active arthritis . The chronic relapsing nature of the disease should be explained to the patients for better compliance with therapeutic modalities.

Conjunctivitis is usually self-limiting. A slit slit – lamp examination is necessary to rule out uveitis , which if present has to be managed with topical corticosteroids, cyclopl a egics and mydriatics. Keratoderma blenorrhagica blennorrhagica is treated using topical steroids and keratolytics. Low potency topical steroids are used in circinate balanitis. [10]

Non Non – steroidal anti anti – inflammatory drugs (NSAID ’ s) are highly effective in pain management in patients with Re A active arthritis . Intra Intra – articular steroids are advocated in oligo/monoarticular disease. The use of systemic steroids has been discouraged except in severe cases where short courses may be given. [15]

Antibiotics are useful in the post post – venereal form of Re A active arthritis . Their role in the post post – dysenteric form remains controversial. Commonly used antibiotics include erythromycin, ciprofloxacin, tetracycline and doxycycline. [11]

In patients who fail to respond to the above mentioned conventional therapy, a more aggressive therapeutic approach is needed. This includes Disease d isease modifying anti anti – rheumatic drugs (DMARD’s) .

References

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