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Review Article

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TO REVIEW THE EFFECT OF RHEUMATOID ARTHRITIS ON EYE

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ABSTRACT

Rheumatoid arthritis (RA) is the most common autoimmune disease. Ocular manifestations of this autoimmune disease vary and are mainly keratoconjunctivitis sicca, episcleritis, scleritis and keratitis. Their appearance, as well as their severity are related to RA chronicity and resistance to therapy. The treatment consists of corticosteroids, NSAIDs and cytotoxic drugs, depending on the type of ocular manifestations and the patient's response to treatment. Rheumatoid arthritis is the most common systemic autoimmune disease, and affects middle age women three time more often than the men in a percentage of 0.5% - 2% of the general population. It is a chronic inflammatory disease characterized by a symmetric sterile and progressive synovitis within joints. The disease is also characterized in 80% of the patients by positive rheumatoid factor but there is also a group of auto- antibodies which are called anti-CCPs and are of great significance for the diagnosis of the disease. Most of the systemic autoimmune diseases have signs and symptoms from the eye and so does Rheumatoid Arthritis. Approximately 25% of patients will have ocular manifestations like scleritis, episcleritis, keratoconjunctivitis sicca, keratitis, corneal disease. retinal vasculitis, episcleral nodules, retinal detachments, and macular oedema.

KEYWORDS: Rheumatoid Arthritis, Ocular Manifestations, Autoimmunity, Nodules, Red Eye, Scleritis, Episcleritis, Keratitis.

INTRODUCTION

Rheumatoid arthritis, or RA, is an autoimmune and inflammatory disease, which means that your immune system attacks healthy cells in your body by mistake, causing inflammation (painful swelling) in the affected parts of the body. RA mainly attacks the joints, usually many joints at once. RA commonly affects joints in the hands, wrists, and knees. In a joint with RA, the lining of the joint becomes inflamed, causing damage to joint tissue. This tissue damage can cause long-lasting or chronic pain, unsteadiness (lack of balance), and deformity. RA can also affect other tissues throughout the body and cause problems in organs such as the lungs, heart, and eyes in which Eye problems are some of the more common complications for people with rheumatoid arthritis, and they can lead to corneal damage and ultimately impair vision if left untreated. Ocular manifestations of RA include episcleritis, scleritis, peripheral ulcerative keratitis and keratoconjunctivitis sicca (KCS) or dry eye.[1] Ophthalmic manifestation as well as signs and symptoms of each condition will be discussed in this review.

AIM

To study the effect of Rheumatoid Arthritis on Eye ball.

OBJECTIVES

- To study details about RA.
- To study the effect of Rheumatoid Arthritis on Eye ball.

MATERIAL AND METHODS

Material related to Rheumatoid arthritis and related to different eye diseases is collected from text book of ophthalmology, medicine, and different Articles.

Review of Rheumatoid arthritis (RA)

Rheumatoid arthritis (RA)

RA is a chronic, progressive autoimmune disease of unknown cause. It is characterized by persistent inflammation that primarily affects the peripheral joints. It usually starts as an insidious symmetrical arthritis and has an unpredictable and variable course, although pain and disability can be minimized if the condition is recognized early and treated promptly and appropriately. According to ayurveda Amavata is the most common endogenous disease which is produced due to frequently formation of Ama in the human body. It is the commonest among chronic inflammatory joint disease in which joints becomes swollen, painful & stiff. Due to its chronicity & complications it has taken the foremost

place among the joint disease. It continues to pose challenge to the physician due to severe morbidity & crippling nature.

Pathophysiology

Pathogenesis involves multiple factors, including both genetic and environmental influences. Immune cells and soluble inflammatory mediators play a crucial role in the pathogenesis, although the relative contribution of individual components remains uncertain. Proliferation of cells in the synovial layer of the joint, together with infiltration by various cell populations, as or chest rated by cytokines, chemokines, growth factors, and hormones, produces a locally invasive pannus that is capable of invading and ultimately destroying cartilage, bone, and surrounding soft tissues.

Effect of Rheumatoid Arthritis on Eye

Scleritis and Episcleritis

Scleritis is a chronic, painful, and potentially blinding inflammatory disease characterized by oedema and cellular infiltration of the scleral and episcleral tissues (outer coat of the eye). Scleritis may be isolated to the eye but is commonly associated with systemic autoimmune disorders, including rheumatoid arthritis, systemic lupus erythematosus, relapsing polychondritis.

Scleritis may be classified into anterior and posterior.[2,3] Anterior scleritis can be diffuse, nodular, necrotizing with inflammation (necrotizing), and necrotizing without inflammation. The most common clinical forms are diffuse scleritis and nodular scleritis. Necrotizing scleritis with or without inflammation is much less frequent, more ominous, and frequently associated with systemic autoimmune disorders and peripheral ulcerative keratitis. Posterior scleritis is characterized by flattening of the posterior aspect of the globe revealed with imaging, thickening of the posterior coats of the eye (choroid and sclera), and retrobulbar oedema.[4]

Signs and symptoms of scleritis[5]

- Redness and swelling of sclera.
- Pain and tenderness in eye.
- Blurred vision.
- Frequent watering (tearing) of eye.
- Sensitivity to light(photophobia).

Treatment

If scleritis is diagnosed, immediate treatment will be necessary. Treatment will vary depending on the type of scleritis, and can include[6]

- Steroid eye drops.
- Anti-inflammation medications - nonsteroidal anti-inflammatories or corticosteroids (prednisone).
- Oral antibiotic or antiviral drugs.

Non-necrotising types are usually treated with oral NSAID medication. If this is not effective, oral steroids are needed. Some doctors treat scleritis with injections of steroid medication into the sclera or around the eye. If these treatments do not work then immunosuppressant drugs such as methotrexate and cyclophosphamide may be used. They can take several weeks to work. Some of the new 'biological agents' such as rituximab can also be effective.

Necrotising scleritis is treated with oral steroids and immunosuppressants as early as possible. Treatment of non-infectious scleritis always requires systemic therapy with nonsteroidal anti-inflammatory drugs (NSAIDs), glucocorticoids, or other immunosuppressive drugs.

Episcleritis

Episcleritis is generally a benign, self-limited inflammation of the episcleral tissues associated with segmental (70%) or diffuse (30%) episcleral redness that is bright red or salmon pink.

There may be unilateral or bilateral involvement and a simple or nodular appearance. It is usually found in patients 20–50 years of age.[7]

Symptoms of episcleritis[8]

- Redness.
- Pain and discomfort in eye.
- Sensitivity to light(photophobia).
- Watering of eye.

Treatment

Most cases of episcleritis are mild, transient, and will resolve without intervention within 2 to 21 days. Supportive treatment with refrigerated artificial tears at least four times daily is a common recommendation. Some patients require medical intervention depending on the severity of their symptoms. For those patients who require prescription medication, a mild topical corticosteroid such as fluorometholone 0.1% or loteprednol etabonate 0.5% may be prescribed four times a day for 1 to 2 weeks then tapered down. Although the risk of steroid response causing ocular hypertension is rare with these mild steroids, patients should follow up 1 to 2 weeks after initiating treatment to monitor intraocular pressure and evaluate for resolution of the episcleritis. If the episcleral inflammation does not respond to fluorometholone 0.1% or loteprednol etabonate 0.5%, then the clinician may need to prescribe prednisolone acetate 1% four times a day. This is a more potent corticosteroid with greater anti-inflammatory effects but also has a higher risk of ocular hypertension. Topical steroids may also cause posterior sub-capsular cataracts and increase a patient's susceptibility for infection; therefore, it is necessary that they are used judiciously.[9]

Topical NSAIDs (diclofenac 0.1% and ketorolac 0.5%) may reduce the mild pain and inflammation associated with episcleritis without affecting intraocular pressure.[10] Oral NSAIDs such as ibuprofen or naproxen may be used as an alternative to topical steroids or if topical steroids do not adequately resolve the inflammation. The

dosage for ibuprofen is 200 to 600mg 3 to 4 times per day, and the dosage for naproxen is 250 to 500 mg twice per day for up to two weeks. Oral NSAIDs should be used with caution because of the risk of gastric ulcers and should be prescribed with an antacid such as omeprazole 20 mg daily or ranitidine 150 mg twice per day.

Uveitis

The uvea is the middle, pigmented layer of the eye that consists of vascular structures including the iris, ciliary body, and choroid. Inflammation of the uvea is referred to as uveitis.[11]

Uveitis can classify as Anterior uveitis, intermediate uveitis, and posterior uveitis.

Anterior uveitis: (also called iritis or iridocyclitis)- Inflammation affecting the iris and the ciliary body. It may be unilateral or bilateral and usually presents with pain, photophobia, and blurred vision, with or without red eye. Symptoms may range from a quiet white eye to a very painful red eye.

Intermediate uveitis: where inflammation of the vitreous humour or pars plana or both occur. It occurs with blurred vision, floaters, redness of eye, and macular oedema.

Posterior uveitis: inflammation affecting the choroid, retina, or both. It is present with floaters, reduced visual acuity, photophobia, blurred or lost vision, difficulty seeing in the dark and difficulty seeing colour.[12]

Anterior uveitis is the most common type and the mean age of onset is 37.2 years with a male-to-female ratio of 1:1.4. Slit lamp bio microscopy demonstrates white blood cells and flare (or protein exudation) in the anterior chamber, cellular deposits on the posterior surface of the cornea (keratic precipitates or KP), and occasionally adhesions (synechiae) between the iris and lens. Hypopyon (or layering of white blood cells in the anterior chamber) is seen.[13]

Treatment

Topical cycloplegics (e.g. atropine or homatropine) to lessen sequalae such as posterior synechiae (attachment of the iris to the lens which may result in decreased vision and pupillary block glaucoma). Dilation of the pupil also relieves pain by relaxing the muscle in your eye. and helps stabilize the blood-aqueous barrier.[14]

In anterior and posterior uveitis -Topical steroids (Corticosteroids) are the drug of choice. Steroids act by modifying and decreasing the inflammatory response in the eye. They inhibit both the cyclooxygenase pathways of inflammatory response and decrease intraocular inflammation. Occasionally subconjunctival, or systemic steroids are needed.

Intermediate uveitis

- Topical therapy (prednisolone acetate 1% or prednisolone sodium phosphate 1%).
- Periocular injections of corticosteroids (dexamethasone, triamcinolone).
- Oral prednisone.
- Intravitreal triamcinolone acetonide injections.[15]

Corneal disease (Keratitis)

Keratitis is an inflammatory condition that affects the cornea of your eye. Peripheral ulcerative keratitis (PUK) is an inflammatory disease which affects the cornea leading to keratolysis and stromal thinning of the peripheral cornea (usually within 2 mm of the corneoscleral limbus). There is commonly an area of vascular hyperaemia in the limbus adjacent to the region of corneal thinning from episcleritis or scleritis. PUK is typically associated with an underlying rheumatologic condition, most commonly rheumatoid arthritis.[19]

Dry Eye syndrome (keratoconjunctivitis sicca)

Dry eyes also known as dry eye disease dry eye syndrome and keratoconjunctivitis sicca, is the most common ocular manifestation of RA. It can occur because of meibomian gland, lacrimal gland, accessory lacrimal gland, or goblet cell dysfunction. Keratoconjunctivitis sicca is chronic, bilateral desiccation of the conjunctiva and cornea caused by too little tear production or accelerated tear evaporation. It is more common in patients over 50 years. It can be associated with dry mouth (Sjögren syndrome) and rheumatoid arthritis.[16]

Symptoms of Dry eye include[17]

- Variable pain and redness.
- Decreased in vision.
- Uni/bilateral peripheral corneal ulceration with epithelial defect.
- Stromal thinning.
- limbal inflammation, and scleritis.
- Diminution in secretion of tears.
- Sensitivity to light (Photophobia).
- A sensation of having something in your eyes.

Treatment[18]

Treatment consists of supportive care to reduce the discomfort include

1. Tear substitutes- Artificial tears remain the mainstay in the treatment of dry eye. These are available as drops, ointment, and slow-release inserts. Mostly available artificial tear drops contain either cellulose derivative (e.g., 0.25 to 0.7%methyl cellulose and 0.3% Hypromellose) or polyvinyl alcohol (1.4%).
2. Topical cyclosporine- (0.05%, 0.1%) Is reported tobe very effective drug for dry eye in many recent studies. It helps by reducing the cell-mediated inflammation of the lacrimal tissue.
3. Topical retinoids- Have recently been reported to be useful in reversing the cellular changes (squamous

metaplasia) occurring in the conjunctiva of dry eye patients.

Symptoms of keratitis include[20]

- Redness of eye.
- Pain and irritation in the affected area.
- Blurred vision.
- Photophobia and inability to open your eye.
- Excessive tearing.

Treatment[21]

If you have a mild case of keratitis, your provider may suggest using lubricant eye drops and letting your eye heal on its own.

Treatment is principally with systemic immunosuppression in collaboration with a rheumatologist.

- Systemic steroids, sometimes via pulsed intravenous administration, are used to control acute disease, with immunosuppressive therapy and biological blockers for longer-term management.
- Topical lubricants (preservative-free).
- Topical antibiotics as prophylaxis if an epithelial defect is present.
- Oral tetracycline (e.g. doxycycline 100 mg once or twice daily) for its anticollagenase effect.

In PUK- immunomodulatory agent is often started concurrently with the corticosteroids. The choice of immunosuppressive medication depends on the underlying systemic disease process and its severity. The first-line for rheumatoid arthritis-associated PUK is often methotrexate (5-25 mg once weekly). If this proves ineffective, other immunomodulatory agents such as cyclophosphamide, azathioprine, cyclosporine, or anti-tumour necrosis factor (TNF) agents may be utilized.

DISCUSSION

RA is associated with many extra articular manifestations, which include ocular diseases such as keratoconjunctivitis sicca, episcleritis, scleritis, peripheral ulcerative keratitis, and retinal vasculitis. These concomitant ocular manifestations are of almost concern and must be addressed because of the high potential for permanent damage and blindness if they are allowed to run their course with- out intervention. Collaborative efforts between the ophthalmologists and rheumatologists involved in the evaluation and treatment of patients with RA are essential to effectively manage any ocular complications that may arise.

CONCLUSION

Rheumatologic disease may manifest as a variety of ophthalmologic conditions with a wide range of signs and symptoms. Such signs and symptoms include ocular burning, itching, redness, pain, photophobia, blurred or decreased vision and warrant a referral for ophthalmologic evaluation. Additionally, many medications used to treat rheumatologic disease, including Plaquenil (hydroxychloroquine), chloroquine, and systemic corticosteroids, may adversely affect the eye and require routine screening. A low threshold for ophthalmologic consultation should exist when evaluating and treating a diversity of rheumatologic disease.[21]

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