The eye in rheumatoid arthritis

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Rheumatoid arthritis (RA) is a systemic inflammatory disease associated with a number of extra-articular organ manifestations. Eye involvement is a frequent finding in patients with RA. Ocular conditions that occur because of RA are keratoconjunctivitis sicca (KCS), episcleritis, scleritis, corneal pathology and retinal vasculitis (Table I). Optic nerve involvement in the form of neuritis or ischaemic neuropathy has been reported and tenosynovitis of the superior oblique muscle sheath (Brown's syndrome) is also an occasional manifestation. It should be borne in mind that the eye may also be affected by the treatment of RA with drugs such as chloroquine, which can cause loss of vision as a result of maculopathy.

In a study[1] where 691 patients with RA were examined (all patients in stages I or II of the disease according to the criteria of the American College of Rheumatology), the most common manifestation of ocular involvement was KCS. Episcleritis was diagnosed in 5.06% of patients and scleritis in 2.06%, with diffuse scleritis in 1 patient and nodular scleritis in 13 patients. There were no patients with posterior scleritis, necrotising scleritis or scleromalacia in this cohort. Keratitis was diagnosed in 11 patients (1.59%) and retinal vasculitis in 3 patients (0.43%). Ocular involvement was found in 27.2% of patients, with women being more affected.

This article focuses on 5 of the more common manifestations.

Keratoconjunctivitis sicca

Also called Sjögren's syndrome, this is a chronic, inflammatory, autoimmune disease characterised by lymphocytic infiltration of the exocrine glands, leading to diminished glandular secretion.[2] Patients with primary Sjögren's syndrome show evidence of dry eyes and dry mouth, whereas those with secondary Sjögren's syndrome also suffer from an autoimmune disease, most commonly RA. Ocular surface involvement is a frequent feature, characterised by the presence of typical symptoms such as ocular burning, foreign body sensation and blurred vision (worsened by prolonged eye use), as well as widespread epithelial damage of the cornea and conjunctiva, leading to redness of the eyes. Punctate staining of the cornea with fluoroscein can be observed in severe cases. Inflammation causes decreased ocular surface sensation, resulting in further decreased tear secretion. The increase in concentration of pro-inflammatory agents on the ocular surface may even lead to sight-threatening corneal complications, including ulceration and perforation. Management includes tear supplements and referral to an ophthalmologist.

Episcleritis

Episcleritis is a self-limiting, benign inflammation of the episclera. Ocular redness with slight tenderness typically lasts days to weeks and resolves spontaneously in most cases.[3] Inflammation is superficial with a bright red or pink colour.

- **Diffuse episcleritis:** Episcleritis may be localised to a sector of the globe in most cases (Fig. 1), but may involve the entire globe in some.

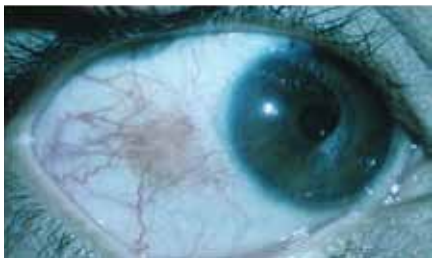

Fig. 1. Episcleritis.

- **Nodular episcleritis:** A localised, mobile nodule may develop in nodular episcleritis.

A work-up for underlying causes should be carried out only in patients with multiple recurrences, as most cases of episcleritis are not associated with underlying systemic disease. Topical or systemic non-steroidal anti-inflammatory drugs (NSAIDs) may be prescribed for pain relief in a few patients.

Scleritis

Scleritis is a more severe inflammatory condition than episcleritis. It is caused by an immune-mediated vasculitis and may take various forms. One-third to two-thirds of patients with scleritis have underlying systemic disease, which may include connective tissue diseases, seronegative spondylo-arthritis and infections. Scleritis causes a boring ocular pain which may wake the patient at night and be referred to the temple or jaw. The inflamed sclera has a 'violaceous hue' when viewed in natural light with injection of the deeper, larger blood vessels.[3]

- **Diffuse anterior scleritis:** A portion (zone) or the entire anterior sclera (Fig. 2) is involved.

Fig. 2. Diffuse scleritis.

- **Nodular anterior scleritis:** An immobile scleral nodule with a deep red to purple colour is found.

- **Necrotising scleritis:** This is the most destructive form of scleritis and patients suffer severe pain. If prompt treatment is not initiated, the inflammation may spread posteriorly and circumferentially, thereby involving the entire globe.

Table I. Ocular manifestations of rheumatoid arthritis

Ocular manifestation	Symptoms	Signs
Keratoconjunctivitis sicca	Burning, foreign body sensation and blurred vision	Redness of conjunctiva with slight corneal haze in severe cases
Episcleritis	Red eye and tender to touch	Sectoral or diffuse superficial injection of globe
Scleritis	Red eye with severe pain	Injection of deeper layers with deep red colour and sometimes associated immobile nodule
Corneal pathology	Red eye with pain and reduced vision	Corneal thinning, ulceration and opacification
Retinal vasculitis	Reduced vision if central area affected	Sheathed retinal vessels with haemorrhage and exudates
Optic neuritis/neuropathy	Profound visual loss	Afferent pupil defect and reduced colour vision
Brown's syndrome	Diplopia	Restricted elevation of globe in adduction

- **Scleromalacia perforans:** In patients with long-standing RA, the sclera becomes extremely thin and develops a blue-grey colour after the inflammation of repeated episodes of necrotising scleritis subsides. The globe may rupture with minimal trauma.

- **Posterior scleritis:** This may occur in isolation or with anterior scleritis. Patients have pain, proptosis, loss of vision, restricted eye movements, disc swelling and even exudative retinal detachment.

Urgent referral to an ophthalmologist is required for most forms of scleritis. Scleritis may be complicated by keratitis, uveitis, cataract and glaucoma. Patients need analgesia, including systemic NSAIDs, and investigation for underlying disease. A work-up should be done in conjunction with a rheumatologist if auto-immune disease is suspected.

Corneal changes

- **Peripheral ulcerative keratitis (PUK):** RA-associated PUK often has a poor visual outcome and its appearance may herald the transformation of a patient's disease into the systemic vasculitic phase.[4] Episodes of keratitis usually correlate with exacerbations of systemic disease activity. There is usually an area of inflammation of the conjunctiva, episclera or sclera adjacent to the area of crescent-shaped peripheral corneal ulceration and thinning. Management should include aggressive immunosuppression if the associated morbidity and mortality are to be avoided. Cell-mediated mechanisms appear to be important in this vaso-occlusive disease and a combination of corticosteroids and cyclosporin is probably the regimen of choice.

- **Peripheral thinning without ulceration ('contact lens' cornea):** This occurs after episodes of PUK result in a thinned, scarred peripheral cornea (Fig. 3).

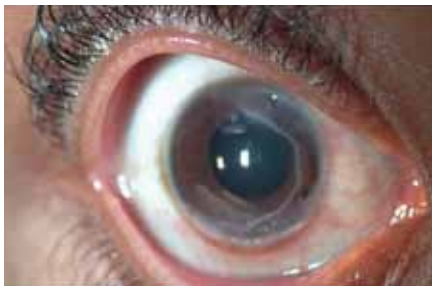

Fig. 3. Peripheral corneal thinning without ulceration.

- **Sclerosing keratitis:** This is characterised by peripheral thickening and opacification of the corneal stroma adjacent to the site of scleritis.

- **Acute stromal keratitis:** Peripheral corneal infiltrates are superficial or mid-stromal and associated with non-necrotising scleritis.

- **Acute 'corneal melt' (keratolysis):** Rapidly progressive thinning may occur in an area of already thinned cornea (peripherally or centrally) and lead to corneal perforation.[5]

All types of keratitis need ophthalmological assessment and the ocular pathology needs to be managed in conjunction with a rheumatologist.

Retinal vasculitis

Inflammation of the retinal vessels is known to occur in RA. This manifests as perivascular infiltrates and sheathing of the retinal vessels. Vasculitis leads to diffuse leakage with resultant exudation, haemorrhage and retinal oedema.[5] Loss of vision occurs if the macula becomes involved or vascular occlusion occurs. Fundoscopy through a dilated pupil should be performed to diagnose vasculitis.

References available at www.cmej.org.za

Upper limb pain syndromes

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This review addresses common questions about upper limb pain that are encountered in practice.

Which anatomical structures account for shoulder pain?

The shoulder complex comprises the glenohumeral, acromiaclavicular, sterno-clavicular, scapulothoracic joints and the subacromial space. Important soft-tissue structures include the capsule, subacromial bursa and rotator cuff muscles – supraspinatus, infraspinatus, teres minor and subscapularis.

What are the causes of referred pain to the shoulder?

Cervical spine pathology is a common cause of referred pain to the shoulder. Symptoms are exacerbated by neck movements and may be accompanied by radicular pain and/or paraesthesia down the arm. Less commonly, but importantly, referred pain may arise from cardiac disease and sub-diaphragmatic pathology.[1,2]

How does one distinguish articular and soft-tissue pathology of the shoulder?

It is important to consider the pattern of limitation of motion. A capsular pattern that implies articular pathology presents as restriction of passive movement, where external rotation is affected more than abduction and internal rotation is the least affected.[3]

Pain or weakness with active movement of the muscles against resistance suggests degeneration or a tear of a contractile structure:

- resisted abduction → supraspinatus tendon
- resisted external rotation → infraspinatus tendon
- resisted internal rotation → subscapularis tendon.

What is shoulder impingement syndrome?

Rotator cuff muscles traverse a narrow space between the acromion and coracoacromial ligament above and the humeral head below. Tendinitis, bursitis or acromioclavicular joint disease compromises the space and impinges the cuff. This is maximal during abduction. It typically produces the painful arc between 70° and 120° of shoulder abduction. Internal rotation of the abducted shoulder also reproduces the symptoms. Treatment includes non-steroidal anti-inflammatory drugs (NSAIDs), physiotherapy and steroid infiltration into the subacromial space. If patients fail a second steroid infiltration, surgery may need to be considered.[4]