- 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 crescentshaped 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 vasoocclusive 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 nonnecrotising 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

MAHMOOD MOOSA TAR MAHOMED ALLY, MB BCh, FCP (SA)

Professor and Head, Division of Rheumatology, Department of Internal Medicine, School of Medicine, University of Pretoria

Correspondence to: M Ally (tar@up.ac.za)

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, sternoclavicular, 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

What is a frozen shoulder?

Marked limitation of shoulder movement as a result of contraction or fibrosis of the capsule of the glenohumeral joint may follow chronic inflammatory arthritis, previous trauma, or prolonged immobilisation or may be idiopathic. The condition is seen more commonly in diabetics and in patients after myocardial infarction or stroke. Patients typically have a history of chronic shoulder pain with inability to lie on the affected side, followed by progressive stiffening of the shoulder in all ranges of motion, especially external rotation. Most cases resolve over a few months after mobilisation exercises and a corticosteroid infiltration. Recovery may be prolonged, taking up to 2 years, especially in diabetics.5

Cervical spine pathology is a common cause of referred pain to the shoulder.

How often is the shoulder joint involved in systemic or generalised arthritic disorders?

The most common inflammatory arthropathy involving the shoulder joint, including the rotator cuff, is rheumatoid arthritis. Shoulder involvement is also seen in seronegative spondyloarthritis (SpA), affecting about 30% of patients with ankylosing spondylitis. Gouty involvement of the shoulder is uncommon. Glenohumeral osteoarthritis is rare and should prompt a search for secondary causes, such as chronic rotator cuff tear, acromegaly, previous trauma or underlying inflammatory arthritis.⁵

What is the Milwaukee shoulder?

This is a condition seen in elderly women due to calcium hydroxyapatite deposition, resulting in an haemorrhagic effusion, severe rotator cuff degeneration and rapidly progressive destructive arthritis of the shoulder joint.⁵

What is enthesitis?

This is inflammation at sites of tendon, ligament, capsular or fascial insertion into bone. It may occur after injury, in repetitive use syndromes and in certain inflammatory conditions such as SpA and HIV-associated arthropathies.⁶

What are the common causes of medial and lateral elbow pain?

Enthesopathy at the origin of the common wrist flexor is characterised by pain at the medial aspect (golfer's elbow) with tenderness just distal to the medial epicondyle and pain that worsens with wrist flexion. Enthesopathy at the origin of the wrist and finger extensors presents with pain and tenderness over the lateral epicondyle exacerbated by resisted wrist extension (tennis elbow). Management may include

steroid infiltration if the patient does not respond to conservative measures.⁷

Which conditions need to be considered in patients with nonspecific distal arm pain in the absence of obvious pathology?

Repetitive strain injury is usually a diagnosis of exclusion, with occupational overuse being an important risk factor. Some consider this condition to be a variant of fibromyalgia, as patients tend to have associated fatigue and sleep disturbances. Conditions such as carpal tunnel syndrome (CTS) and a small ganglion within the wrist need to be excluded.⁵

What are the signs of carpal tunnel syndrome?

Typically, patients present with sensory loss or paraesthesia along the radial aspect of the ring, middle and index fingers and the thumb. Symptoms may be reproduced by tapping over the carpal tunnel (Tinel's sign) or by full flexion of the wrist for 60 seconds (Phalen's sign). Thenar atrophy may be present, implying chronicity. Predisposing conditions are inflammatory arthropathy, acromegaly, diabetes, hypothyroidism, overuse syndromes and pregnancy.^{7,8}

What is De Quervain's disease?

This is tenosynovitis of the first dorsal compartment of the wrist, which presents with pain on the radial side of the wrist and the base of the thumb. Pinching or grasping movements of the thumb produce pain. A clinical test – Finkelstein's manoeuvre – is positive if pain is reproduced along the first dorsal compartment, when the wrist is moved in an ulnar direction with the thumb clasped in the palm. Local steroid infiltration into the tendon sheath may be required if conservative measures with NSAIDs and wrist/thumb splint fail.^{7,8}

What is trigger finger?

This is tenosynovitis affecting the flexor tendons of the fingers or thumb, resulting in fibrosis or nodule formation. It interferes with the normal smooth movement of the tendon, 'catching' or locking as the tendon passes under the A1 pulley in the region of the metacarpophalangeal joint. Patients may have tenderness at the base of the finger with a palpable nodule. Pain is exacerbated by stretching the tendon in extension or resisted flexion.

Patients may benefit from local steroid infiltration. Conservative measures include immobilisation for 4 - 6 weeks, with a repeat steroid infiltration; if symptoms persist for more than 6 weeks surgery should be considered.^{7,8}

Summary

 Upper limb pain syndromes may be due to a wide variety of local or systemic factors, many of which may be effectively managed in primary care. Due consideration has to be given to referred pain so as not to miss pathology elsewhere.

References available at www.cmej.org.za

Fibromyalgia

NEETA PATEL, MB ChB, FCP (SA)

Principal Specialist and Lecturer, Department of Rheumatology, Nelson R Mandela School of Medicine, University of KwaZulu-Natal and Inkosi Albert Luthuli Central Hospital, Durban

GIRISH M MODY, MB ChB, FCP (SA), MD, FRCP (Lond), Fellow of the University of KwaZulu-Natal

Aaron Family Professor and Head of the Department of Rheumatology, Nelson R Mandela School of Medicine, University of KwaZulu-Natal and Inkosi Albert Luthuli Central Hospital, Durban

Correspondence to: N Patel (pateln2@ukzn.ac.za)

Over the past decade there have been major advances in our understanding of fibromyalgia (FM). The identification of alterations in the levels of many neurotransmitters has resulted in newer targets for therapeutic intervention in this condition.

FM is a chronic disorder characterised by widespread musculoskeletal pain. According to the American College of Rheumatology (ACR) fibromyalgia classification criteria (1990)¹ patients must have a history of widespread pain that has been present for at least 3 months. The pain can be elicited on digital palpation by manual pressure of