

agent (probenecid), or allopurinol desensitisation (only in cases of mild rash). Note that probenecid is contraindicated in kidney disease and when a history of kidney stones is present. The daily colchicine may be discontinued when the target uric acid levels have been reached.<sup>9</sup> Colchicine may then be given as needed for an acute attack at doses of 0.5 mg three times daily until the attack subsides. NSAIDs may also be given, as well as corticosteroids (systemic or intra-articular). It is important to note that corticosteroids have many adverse effects and are often misused to the detriment of the gout patient. It is better to continue the allopurinol during an acute attack rather than discontinuing the drug. This will lead to less fluctuation of drug levels and better control of the disease.

Management of co-morbid conditions in the gout patient is important. There is a higher risk of atherosclerosis and patients are prone to have hypertension, glucose intolerance and hypercholesterolaemia. These conditions should be treated aggressively. Hypertension may be resistant to treatment and it is not always possible to discontinue diuretic therapy. A useful drug for hypertension that increases the urine excretion of uric acid is losartan.<sup>10</sup> The key to successfully managing gout is early and aggressive treatment that includes education, motivation and lifestyle modification.

#### In summary

- Gout is strongly modified by the lifestyle of a patient. Education, motivation and lifestyle modification play a major role in treatment.
- Gout progresses through different stages that are related to the total amount of uric acid that accumulates in the body over time.
- The eventual outcome in the poorly managed patient is polyarticular joint damage, functional impairment, nephropathy and cardiovascular complications.
- Treating acute attacks only will not prevent the eventual outcome of the disease.
- Effective management of gout should aim to prevent joint damage and systemic complications by lowering and maintaining the uric acid levels to a target of below 0.35 mmol/l.
- Corticosteroids are often used without considering the detrimental side-effects and do not lower uric acid levels, while contributing to worsening the co-morbid diseases that frequently occur in patients with gout.
- Xanthine oxidase inhibitors are effective in under-excretors and over-producers by decreasing uric acid levels and lightening the load of uric acid to be excreted.
- Xanthine oxidase inhibitors should not be discontinued in the event of an acute attack in the patient who is on treatment.

- Probenecid should be used with caution in patients with a history of kidney stones and nephropathy.
- Many drugs influence the excretion of uric acid and may precipitate acute attacks, while losartan is a good choice in the management of hypertension because of its uricosuric characteristics.

References available at [www.cmej.org.za](http://www.cmej.org.za)

### Lower limb pain syndromes

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Lower limb pain is a common presenting complaint in primary care with aetiological factors such as trauma, repetitive strain injury and systemic inflammatory disorders. This review focuses on some commonly encountered problems when dealing with patients who present with lower limb pathology.

#### What are the common causes of referred or radicular pain in the lower limb?

Intra-abdominal, gynaecological, urological and lumbar spine pathology may be referred to the lower limbs. Lumbar spine pathology is a common cause of radicular pain, with disc herniation at L4 - L5 or L5 - S1 being the most frequent levels of involvement. Sensory loss and paraesthesia in the typical dermatomal pattern, or loss of reflexes (L3 - L4: loss of patellar tendon reflex or S1: loss of Achilles tendon reflex)<sup>1</sup> may be clinically evident.

#### What are the causes of hip pain in children and adolescents?

Participation in sports and related activities needs to be considered in this age group. Labral tears, in particular, present typically with anterior hip pain or groin pain. There may be associated symptoms of clicking or locking of the hip. One of the most common problems in young patients is a slipped capital femoral epiphysis. The patient usually complains of pain in the groin, thigh or knee with limitation of external rotation and leg length shortening. Idiopathic avascular necrosis (AVN) of the femur (Legg-Calvé-Perthes disease) may occur in children,

commonly boys between the ages of 4 and 9 years.<sup>2</sup>

#### Causes of avascular necrosis of the femoral head in adults

Systemic steroids and alcohol are well-recognised causes of AVN in adults, but conditions associated with hypercoagulable states such as systemic lupus erythematosus, antiphospholipid syndrome, sickle cell disease, primary coagulopathies and metabolic disorders such as diabetes need to be considered. A high index of suspicion is needed for the early detection and diagnosis of AVN as initial clinical features are nonspecific, with insidious onset of progressive hip pain exacerbated by weight bearing with normal range of motion.<sup>3,4</sup>

#### How do you diagnose and treat bursitis around the hip?

Two important bursae may lead to hip pain: iliopsoas and greater trochanteric bursa. The iliopsoas bursa lies between the iliopsoas muscle/tendon and the pubic eminence. Typically patients have pain and tenderness in the groin with pain on resisted flexion of the hip. The more common bursal lesion is trochanteric bursitis. Patients have difficulty lying on the affected side with localised tenderness over the greater trochanter. They also have pain on adduction and passive internal rotation of the hip. The ischial bursa lies over the ischial tuberosity in close proximity to the sciatic nerve. Common presenting symptoms include buttock pain worsened by sitting and associated paraesthesia down the back of the leg. Treatment may include non-steroidal anti-inflammatory drugs (NSAIDs), analgesia and local steroid infiltration under ultrasound guidance for the deep bursae.<sup>3,4</sup>

### What are the extra-articular causes of knee pain?

Anatomical structures with sensory nerve supply include the patellar tendon, retinacular tissue, bursa, synovial tissue and the fat pads. Patellar and quadriceps tendinopathy may be caused by mechanical factors, overload or enthesitis. Inflammation in the infrapatellar fat pad (Hoffa's fat pad) presents with anterior knee pain below the pole of the patellar with exacerbation of the symptoms by knee extension.

A common cause for anteromedial pain is inflammation of the pes anserine bursa, which separates the medial collateral ligament and the medial hamstring tendons. Pain is worse on knee extension, resisted knee flexion and/or adduction of the legs.<sup>5</sup>

### What is the 'too many toes' sign?

The posterior tibial tendon is responsible for the suspension of the medial arch of the foot. Dysfunction of the tendon either from degenerative changes or inflammatory disorders results in an acquired flat foot with valgus hindfoot and forefoot abduction. If the foot is inspected from behind, more toes are visible lateral to the ankle than will be seen in a normal foot.<sup>6</sup>

### What are the risk factors for Achilles tendon rupture?

The three common sites of rupture are the hypovascular region of the tendon, which can be found 2 - 6 cm proximal to the insertion, the enthesial site and the musculotendinous junction. Enthesitis from inflammatory rheumatic diseases such as seronegative spondyloarthropathies, rheumatoid arthritis and gout predisposes the patient to tendon rupture. Other causes of rupture may include steroid treatment, particularly local administration of steroid, xanthomas and oral fluoroquinolones.<sup>6</sup>

### What are the soft-tissue causes of forefoot pain?

One of the most common soft-tissue masses of the foot are the ganglia, often located dorsal to the metatarsophalangeal joints and tendons. Bursitis may occur in the intermetatarsal areas or beneath the metatarsal heads secondary to repetitive trauma, infection or inflammatory disorders.

Sharp burning pain in the ball of the foot usually located between the 3rd and 4th metatarsal heads is most likely due to a Morton's neuroma. This is not a tumour but is related to peripheral fibrosis following compression of the interdigital nerves. Therapy includes changing footwear, orthotics and steroid infiltration.<sup>6</sup>

### Summary

- Lower limb pain is a common presenting complaint with numerous aetiological factors.

- Due consideration of mechanical, traumatic and repetitive strain disorders as well as a search for metabolic and inflammatory conditions will allow for early diagnosis and management.

References available at [www.cmej.org.za](http://www.cmej.org.za)

## The lungs in the rheumatic diseases

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There are many different approaches to the evaluation of the lungs in patients with collagen vascular diseases. Many books have been published about the evaluation of the lungs in patients with rheumatic diseases (connective tissue diseases (CTDs) and rheumatoid arthritis (RA)). Unfortunately, it still remains a challenging task – even for a pulmonologist. As with all medical conditions, there is no substitute for a thorough history and careful examination of the patient. Although significant heterogeneity exists among the different collagen vascular diseases, they share one aspect that is common to their pathogenesis: circulating auto-antibodies and immune-mediated organ dysfunction.

There are several tools to evaluate the lungs of patients with a CTD. Currently none is as valuable as the chest radiograph as an initial screening tool to exclude major pathology. If pathology is identified, a high-resolution computerised tomography (HRCT) scan of the chest is performed to confirm the pathology and ascertain a possible aetiology.<sup>1-3</sup> The most important rheumatic diseases that affect the lungs include:

- RA
- systemic lupus erythematosus (SLE)
- systemic sclerosis
- Sjögren's syndrome
- polymyositis /dermatomyositis /anti-synthetase syndromes
- mixed CTDs
- primary vasculitic syndromes (e.g. Wegener's granulomatosis).

When evaluating the lungs of patients with a CTD several different aspects of the disease process need to be considered before the clinician can come up with an appropriate differential diagnosis. The most important questions the treating clinician (general practitioner, general physician, rheumatologist or pulmonologist) has to consider include:

- Has the patient's CTD been well characterised clinically, serologically and radiologically?
- Is the collagen vascular disease well established and is the patient on appropriate therapy?
- Is there a super-added infective aetiology (i.e. viral, fungal, bacterial or mycobacterium)?
- Is there a possibility of drug toxicity (i.e. methotrexate (MTX) lung toxicity, salazopyrine toxicity or disease related to biological agents)?
- Has the patient developed an unrelated separate pathology (i.e. hypersensitivity pneumonitis, a secondary malignancy or an aspiration pneumonitis)?<sup>4,5</sup>

With the above possibilities in mind, Table I highlights the most common disease-associated (CTD) pulmonary manifestations.

The ILDs pose the greatest diagnostic challenge and therapeutic dilemma. There are various different histological sub-classifications of the ILDs, but the most important include:

- UIP = usual interstitial pneumonia (formerly called CFA= cryptogenic fibrosing alveolitis)
- NSIP = nonspecific interstitial pneumonitis
- OP = organising pneumonia (formerly called BOOP= bronchiolitis obliterans organising pneumonia)
- LIP = lymphocytic interstitial pneumonia.

NSIP is the most common histological pattern associated with a CTD. However, in RA a UIP pattern is more common, while in Sjögren's syndrome LIP is the predominant histological pattern. Furthermore, patients with scleroderma are particularly prone to developing pulmonary vascular hypertension.