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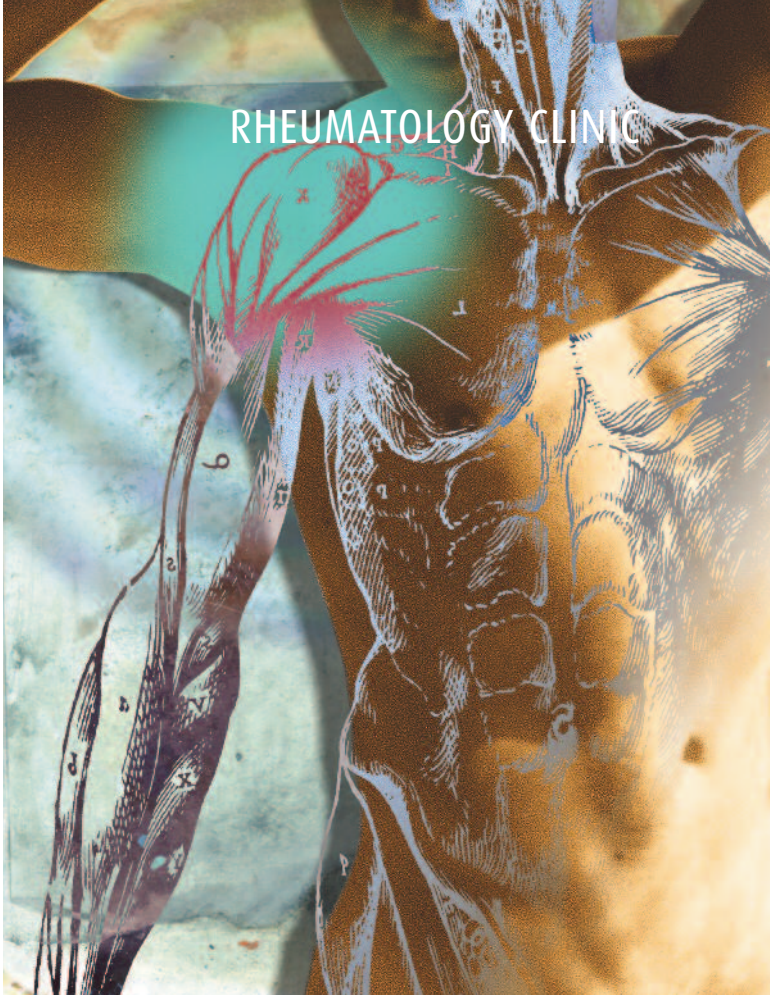

Soft tissue rheumatic disorders

Part 1. Aetiology and diagnosis

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In soft tissue rheumatic disorders, it is important to distinguish noninflammatory causes, such as biomechanical abnormalities or tendon changes with ageing, from inflammatory causes, such as systemic rheumatic or other diseases.

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The General Practice Activity in Australia 2007-08 report estimated that musculoskeletal problems are the fourth most common reason for consultations in general practice (behind respiratory, general and cardiovascular conditions) and account for about 17 of every 100 patient encounters.[1]

In referring to these problems, patients often ask what is the difference between arthritis and rheumatism? Simplistically, we might apply the term 'arthritis' to articular complaints pertaining to the joint and its capsule and reserve the terms 'rheumatism' or 'rheumatic disorder' for complaints of the adjacent soft tissues, the subject of this series of two articles.

Soft tissue rheumatic disorders are ubiquitous. Here, we will outline how the soft tissues respond to ageing, biomechanical problems, overuse injury, systemic rheumatic diseases and other conditions, such as diabetes. We will discuss a conceptual and practical approach to diagnosing soft tissue rheumatic disorders. In the second part of this two-part series in a forthcoming issue, we will outline common local and regional problems and the principles of their treatment.

ANATOMY OF THE JOINT SOFT TISSUES

A review of the microanatomy of the tissues adjacent to the joint may help us understand the wide range of structures that can be affected and potential causes of soft tissue rheumatic disorders. The musculotendinous unit comprises tendon and ligament insertions into bone (entheses), the tendons and ligaments

DEFINITIONS OF TENDON DISORDERS

Tendinopathy: any pathology affecting tendons

Tendinitis: tendinopathy with inflammation (often incorrectly used interchangeably with tendinopathy)

Tendinosis: a noninflammatory condition affecting the tendons

Tenosynovitis: any inflammatory change in the tendon sheath

themselves, associated structures such as the tendon sheaths and bursae, and the interfaces between tendons and muscles.

Entheses

Entheses connect tendons and ligaments to bone and function to dissipate load. They may fail mechanically and may also be the site of inflammation in spondyloarthropathies.

Tendons

Tendons, which attach muscle to bone, are principally composed of type 1 collagen and elastin in a water–proteoglycan matrix manufactured by tenocytes (tendon fibroblasts). The collagen fibres in tendons vary in number and orientation depending on the site, function and muscle attachments of the tendon. For example, the Achilles tendon is markedly thicker and stronger than the finger flexor tendons, reflecting their different functions.

Tendons have a slow intrinsic metabolic rate and can tolerate the low oxygen concentrations and mechanical tension that occur during daily life. However, the corollary is a low capacity to recover after activity and injury. After the skeleton reaches maturity, tenocytes decline in both number and metabolic activity. With ageing, micro-injuries to the tendons increase, and many apparently acute injuries are actually cumulative. These are often simplistically explained as 'wear and tear', but 'wear and defective repair' seems more accurate, as tendinopathy due to collagen disarray and separation is the result of repetitive microtrauma and a failed healing response.

Definitions of tendon disorders are shown in the box on this page (top left). In tendinosis, the noninflammatory form of tendinopathy, histological examination shows neovascularisation and increased protein synthesis, with little or no inflammation. Tendinosis often takes many months to improve. Ageing increases the risk of any tendinopathy, as do smoking, obesity and certain medications, such as fluoroquinolones.

Tendon sheaths and bursae

Tendon sheaths are lined by synovium to allow the tendons to glide without friction. Bursae, which are located between anatomical structures that move in relation to one another, are also lined by synovium and similarly exist to reduce friction. Bursae can be conceptualised as sack-like structures and may communicate with joints. Both tendons and bursae are potentially affected by joint pathologies.

AETIOLOGY OF SOFT TISSUE RHEUMATIC DISORDERS

Soft tissue rheumatic disorders can be a consequence of the normal changes of ageing but can also result from systemic rheumatic disease, systemic metabolic disease, overuse injury and medications. The most important differential diagnoses in tendinopathy are summarised in the box on this page (top right).[2] Clues that suggest a systemic rheumatic disease are outlined in the box on the next page.

DIFFERENTIAL DIAGNOSES OF TENDINOPATHY

Primary tendinopathy

- Wear and defective repair

Secondary tendinopathy

Causes may be:

- Metabolic – gout, pseudogout or hydroxyapatite deposition. Crystal deposition in a joint or surrounding tissues can lead to inflammation and pain. The most common cause is gout, caused by deposition of urate crystals
- Diabetic cheiroarthropathy – diabetic joint disease associated with excessive collagen deposition in the hand, limiting joint mobility[2]
- Infective – septic bursitis or tenosynovitis
- Inflammatory – spondyloarthritis, reactive arthritis, inflammatory arthritis and, rarely, systemic lupus erythematosus
- Toxic – fluoroquinolone tendinopathy

HISTORY TAKING

In patients presenting with symptoms related to the soft tissues of the joint, the aim of history taking is:

- to localise the region and structure involved
- to identify whether the complaint is:
  - noninflammatory and related to biomechanical abnormalities or the consequences of tendon pathophysiology with ageing, or
  - inflammatory and potentially related to systemic rheumatic disease or other causes.

The history should include:

- onset of the complaint
- its relationship to recent activities
- current provoking and relieving factors
- any aspects that might give clues as to the underlying cause.

For example, the development of heel pain unrelated to a recent change in exercise in a younger man with psoriasis suggests possible spondyloarthritis. In contrast, the insidious development of heel pain in an overweight, flat-footed person aged 55 years implies a

CLUES TO AN UNDERLYING SYSTEMIC PATHOLOGY

- Multiple sites of tendinopathy or enthesopathy
- Psoriatic lesions or previous diagnosis of psoriasis
- Inflammatory symptoms (e.g. early morning stiffness)
- Extra-articular manifestations of rheumatic disease (e.g. uveitis)
- Global spinal stiffness in an older person – diffuse idiopathic skeletal hyperostosis (DISH or Forestier's disease)
- Diabetes (type 1 or type 2)
- Family history

degenerative cause, such as plantar fasciitis. Achilles tendon pain in a teenage basketballer is more likely to be apophysitis (Sever's disease, caused by overuse and repetitive microtrauma of growth plates of the calcaneus) than a true tendinitis, and clearly differs from a cumulative tendon injury developing in a sedentary person training on weekends for a half marathon.

A systems review should cover features that would suggest or exclude the differential diagnoses in the box on the previous page and clues to systemic pathology in the box on this page.

PHYSICAL EXAMINATION

The aim of a physical examination is to identify the structure affected, to exclude systemic disease and to identify any biomechanical factors that predispose to soft tissue failure. It is not sufficient to examine the affected area. Regional biomechanics should also be assessed and any clues in the history should be followed up to identify conditions such as spondyloarthritis, gout and diffuse idiopathic skeletal hyperostosis (DISH or Forestier's disease).

It should be appreciated that a physical examination, similar to other investigations, has a sensitivity and specificity, and a systematic approach is most likely to identify the cause of symptoms. Details of systematic approaches to physical examination can be found elsewhere.[3,4]

Top tips for examination are outlined below.

- Consider the underlying anatomy. Where is the tenderness located? Resort to the anatomy atlas if in doubt!
- Are there problems with the local biomechanics? For example, does the patient with plantar fasciitis have flat feet and tight calf muscles?
- Conduct a general examination. For example, is psoriasis present? This may not be apparent unless the scalp and less obvious locations such as the natal cleft are examined, and the nails are assessed for pitting and other changes. Always consider the need for a gait, arms, legs and spine (GALS) screen.[5] Any system of examination is better than no system.
- Do not default to expensive imaging as a substitute for a thorough examination.

INVESTIGATION

Sophisticated and accurate musculoskeletal imaging techniques (e.g. ultrasound and MRI) can confirm or refute the possible diagnoses suggested by history and examination. However, imaging should not be used indiscriminately, without reference to history or physical findings. Indiscriminate imaging is not only cost-ineffective but, given that the degenerative changes of ageing are ubiquitous and often do not cause symptoms, it can lead to false-positive results and inappropriate treatments.

CONCLUSION

Soft tissue rheumatic disorders are common. An understanding of the anatomy and biology of tendons, regional biomechanics and the various conditions that can manifest with problems in the tendons, tendon sheaths and bursae can simplify the assessment and investigation of these disorders. In the next article in this two-part series we will focus on specific soft tissue joint problems that illustrate the general approach to diagnosis and treatment. MT

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