Rheumatic Diseases Presenting as Sports-Related Injuries

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Abstract

Most individuals seeking consultation at sports medicine clinics are young, healthy athletes with injuries related to a specific activity. However, these athletes may have other systemic pathologies, such as rheumatic diseases, that may initially mimic sports-related injuries. As rheumatic diseases often affect the musculoskeletal system, they may masquerade as traumatic or mechanical conditions. A systematic review of the literature found numerous case reports of athletes who presented with apparent mechanical low back pain, sciatica pain, hip pain, meniscal tear, ankle sprain, rotator cuff syndrome and stress fractures and who, on further investigation, were found to have manifestations of rheumatic diseases. Common systemic, inflammatory causes of these musculoskeletal complaints include ankylosing spondylitis (AS), gout, chondrocalcinosis, psoriatic enthesopathy and early rheumatoid arthritis (RA).

Low back pain is often mechanical among athletes, but cases have been described where spondyloarthritis, especially AS, has been diagnosed. Neck pain, another common mechanical symptom in athletes, can be an atypical
presentation of AS or early RA. Hip or groin pain is frequently related to injuries in the hip joint and its surrounding structures. However, differential diagnosis should be made with AS, RA, gout, pseudogout, and less often with haemochromatosis and synovial chondromatosis. In athletes presenting with peripheral arthropathy, it is mandatory to investigate autoimmune arthritis (AS, RA, juvenile idiopathic arthritis and systemic lupus erythematosus), crystal-induced arthritis, Lyme disease and pigmented villonodular synovitis. Musculoskeletal soft tissue disorders (bursitis, tendinopathies, enthesitis and carpal tunnel syndrome) are a frequent cause of pain and disability in both competitive and recreational athletes, and are related to acute injuries or overuse. However, these disorders may occasionally be a manifestation of RA, spondyloarthritis, gout and pseudogout.

Effective management of athletes presenting with musculoskeletal complaints requires a structured history, physical examination, and definitive diagnosis to distinguish soft tissue problems from joint problems and an inflammatory syndrome from a non-inflammatory syndrome. Clues to a systemic inflammatory aetiology may include constitutional symptoms, morning stiffness, elevated acute-phase reactants and progressive symptoms despite modification of physical activity. The mechanism of injury or lack thereof is also a clue to any underlying disease. In these circumstances, more complete workup is reasonable, including radiographs, magnetic resonance imaging and laboratory testing for autoantibodies.

In daily practice, sports medicine practitioners see a variety of sports-related injuries. There is a temptation to attribute a mechanical diagnosis to every patient who presents with a joint complaint. However, it is necessary to maintain an index of suspicion for inflammatory joint diseases, especially rheumatic diseases. This article presents a literature review of rheumatic diseases presenting as sports-related injuries in five common conditions: low back pain, neck pain, hip/groin pain, peripheral arthropathy and soft-tissue disorders.

1. Low Back Pain

Low back pain is a common presenting complaint for the general population. Reported lifetime prevalence varies from 49% to 70% and point prevalences from 12% to 30% are reported in Western countries.\[1\]

Low back pain is common in athletes, with its prevalence estimates ranging from 1% to >30%.\[2,3\] Low back pain is one of the most common reasons for missed playing time in professional athletes, yet the prevalence among recreational athletes is not well known. In the athletic population, the majority of low back pain is mechanical and is thought to be related to muscle strain or sprains of the ligamentous structures of the lower back. Disc herniation, compression fracture, spinal stenosis, and degenerative disease can occur in the mature athlete. In young athletes, the most common diagnosis is spondylolysis.\[4\]
It is interesting to note that disorders simulating athletic injury, including tumours and inflammatory connective tissue disease, may be encountered.\[^5\] It has been estimated that about 5% of patients with chronic low back pain seen in the primary care setting are classified as having spondyloarthritis.\[^6\] Another study in the US found that of all patients with back pain in primary care clinics, 0.3% have a diagnosis of ankylosing spondylitis (AS).\[^1\]

AS is a chronic inflammatory disease that belongs to the group of diseases called spondyloarthritis. Besides AS, which is the most frequent, spondyloarthritis comprises reactive arthritis or Reiter’s syndrome, arthritis/spondylitis associated with inflammatory bowel disease (enteropathic arthritis), arthritis/spondylitis with psoriasis or psoriatic arthritis, and undifferentiated spondyloarthritis. The leading clinical symptoms for all subsets of spondyloarthritis are inflammatory back pain and/or asymmetrical arthritis, predominantly of the lower limbs.\[^6\] The European Spondyloarthropathy Study Group published a study aimed at developing classification criteria for the entire group of spondyloarthritis, with the specific intention of including patients with undifferentiated spondyloarthritis. The following classification criteria for spondyloarthritis were proposed: inflammatory spinal pain or synovitis (asymmetric or predominantly in the lower limbs), together with at least one of the following: positive family history, psoriasis, inflammatory bowel disease, urethritis, acute diarrhoea, alternating buttock pain, enthesopathy, or sacroiliitis as determined from radiography of the pelvic region.\[^7\]

Inflammatory low back pain is the hallmark of AS and is defined as pain associated with significant stiffness (especially morning stiffness for >1 hour), present for at least 3 months’ duration that improves with exercise, but is not relieved by rest.\[^8\] Axial manifestations are seen less frequently in the other diseases and occur in 40% of patients with reactive arthritis, 10% in those with inflammatory bowel disease, and only 5% in persons with psoriatic spondyloarthritis.\[^6\]

Evaluating the incidence of AS in athletes, Wordsworth and Mowat\[^9\] performed a review of 100 patients with AS and found that 61% of them had participated in regular athletic activity in their youth. The authors found that 30 of 45 male AS patients who exercised regularly had to reduce their activity level at an average age of 23 years. In contrast, the 30 healthy controls in this study continued to engage in their sports to an average age of 29 years. The most frequent initial symptoms of the patients with AS were low back pain (41%) and sciatica-like pain (25%). Given these symptoms, a physician might easily initially mistake AS for a sport-related injury.

It has become increasingly evident that in many patients with AS, it takes years from the onset of inflammatory low back pain until the appearance of radiographic sacroiliitis. This is especially true in women, who may never develop radiographic changes. For example, Dick Tayler, a winner of the 10 000-m race at the 1974 Commonwealth games, had been continually plagued by low back pain and Achilles tendon disorders. He was later diagnosed as having AS, the consequences of which forced him to stop running.\[^10\]

The absence of radiographic sacroiliitis in the early stage of disease does not necessarily indicate absence of inflammation in the sacroiliac joint or other parts of the axial skeleton. Recent application of magnetic resonance imaging (MRI) techniques have demonstrated (and confirmed) that ongoing active inflammation does in fact occur in the sacroiliac joint or the spine prior to its appearance on plain radiographs. In light of this, some authors have proposed new or revised criteria to allow the early diagnosis of AS, especially since more effective treatment options have become available. New or revised criteria may comprise all parameters relevant to axial spondyloarthritis including inflammatory back pain, heel pain (enthesitis defined as the inflammation of the enthuses, which are any point of attachment of skeletal muscles or ligaments to bone), peripheral arthritis, dactylitis, acute anterior uveitis, family history of spondyloarthritis, good response to NSAIDs, elevated acute-phase reactants, human leucocyte antigen B27 (HLA-B27), sacroiliitis...
Until recently, the options available to clinicians for treating AS have been limited, with patient education, physical therapy, and NSAIDs being the mainstay of effective therapy. The advent of tumour necrosis factor-α (TNFα) antagonists represents a breakthrough in treating AS. Etanercept, infliximab and adalimumab, the three TNFα antagonists currently approved for the treatment of AS, have been demonstrated as being rapid and consistently effective in reducing the axial and peripheral symptoms and improving patients’ ability to function and their quality of life.[12,13]

The physician assisting young athletes with low back pain with inflammatory characteristics must take into consideration the differential diagnosis with spondyloarthritis, especially AS. A high index of suspicion, early diagnosis and prompt treatment are crucial for slowing disease progression and enabling the athlete to maintain the greatest physical function.

2. Neck Pain

Although the most common causes of neck pain are mechanical, the differential diagnosis for neck pain in athletes may be quite extensive and difficult. One should consider atypical cases of early rheumatoid arthritis (RA) and AS that may present with neck pain as the main symptom and when the symptoms appear to be more inflammatory than typically seen with soft-tissue or degenerative disorders.

The polyarthritis of RA affects the joints of the spine, and particularly the upper cervical spine.[14] Several studies of patients with RA suggest that the cervical spine becomes involved early in the course of disease, often within the first 2 years following the diagnosis. However, rheumatoid involvement of the cervical spine is often asymptomatic.[15] Patients with obvious arthritis in the hands are at increased risk for symptomatic cervical spine abnormalities. When the disease is unquestionably established, cervical spine radiographic abnormalities may include atlantoaxial (C1–2) subluxation, superior migration of the odontoid, subaxial arthritis, and collapse of the lateral masses of C1 from erosion at the facet joints.[16]

Patients with diagnosed RA are at increased risk of spinal cord injury while participating in collision sports because of the increased incidence of cervical instability. Cervical subluxation may be found in 15% of RA patients within 3 years of diagnosis; 17% of RA patients with radiographic abnormalities have neurological symptoms.[17] Thus, a high index of suspicion is important for RA diagnosis when athletes complain of inflammatory neck pain especially with peripheral joint involvement.

As cited above, spondyloarthritis, especially AS, usually affects men and starts in the lumbar spine and sacroiliac joints and subsequently spreads up to the thoracic and cervical spine. However, the disease may present with neck pain as the first manifestation without lower back pain or tenderness of sacroiliac joints, especially in women. Clinically, AS may present as gradual neck pain, stiffness and deformity that can cause great difficulty in performing everyday tasks and lead to a predisposition to fracture, dislocation and atlantoaxial subluxation.[18] Because of the mass fusion process of AS and altered biomechanical state, these patients are at high risk for sustaining spinal fractures that frequently occur after minor trauma and occasionally after no apparent or identifiable trauma. They are common in the lower cervical spine and occur frequently through the intervertebral disc.[19,20] There have also been case reports of cervical fracture in patients with AS following chiropractic manipulation.[21]

3. Hip and Groin Pain

Hip or groin pain seems to occur frequently in sports involving twisting and turning, such as football, soccer, ice hockey, or basketball as well as in sports such as running with repetitive impact.[22] Pain may originate from the hip joint and its surrounding structures, as is seen with a labral tear, osteochondral defect, hip-joint synovitis, stress fractures of the femoral neck, and trochanteric bursitis. It also may arise from the adductor muscles where chronic muscle strain or tendinopathy
occur. Injury to the pubic bones may result in a pubic ramus fracture or osteitis pubis and can be the cause of a patient’s symptoms. Likewise, the lower abdominal muscles may be implicated in iliopsoas strain, rectus abdominis tendonopathy, or sports hernia. The lower thoracic spine, lumbar spine, and sacroiliac joint may refer pain to the groin. However, less common causes of pain in this region, such as some rheumatic disorders, must be considered.[23] The rheumatic diseases that can present as arthritis of the hip are spondyloarthritis (especially AS), RA, gout and pseudogout.

Lovell[23] reviewed the case notes of 189 athletes with chronic groin pain. Diagnoses were determined following a review of their history, clinical examination, local anaesthetic infiltration, radiological investigation, surgical exploration, and clinical progress. The most common pathology was incipient hernia (50%). Other common diagnoses were adductor lesions, osteitis pubis, pubic instability, iliopsoas injuries, spinal nerve compression (referred to groin), rectus abdominis tendonopathy, and stress fractures. Only one patient was diagnosed as having AS, as he presented with sacroiliitis on a bone scan and tested positive for the HLA-B27 antigen.

An example of a difficult and rare diagnosis of the hip and groin pain was described by Doward and co-workers.[24] They reported a case of a 34-year-old Olympic-calibre cyclist who presented with a 1-year history of progressive left hip and groin pain. Her symptoms initially began when she was running, but progressed to the point where they occurred with walking, cycling and lying on the hip. Clinical examination revealed moderately decreased internal rotation, external rotation, forward flexion, and abduction of the left hip compared with the right; and subjective complaints of deep hip pain with internal or external rotation. A radiograph of the left hip revealed slight hip joint narrowing centrally. An MRI arthrogram revealed a small anterior labral tear and innumerable small intermediate-intensity filling defects situated diffusely within the joint fluid thought to be consistent with extensive reactive synovitis in the left hip. Arthroscopic removal of loose bodies was performed and operative evaluation revealed no real labral tear, but damage at the labral cartilaginous junction anteriorly, some damage to the femoral head, and hundreds of cartilaginous loose bodies within the joint. After histological examination, the diagnosis of synovial chondromatosis was made. Seventeen months after surgery, this patient was able to return to her previous athletic activities. The authors suggested that with the increased awareness of labral tears as a source of hip pain in athletes it is important for physicians to keep other causes of hip synovitis in mind.

McCurdie and Perry[25] reported two cases of haemochromatosis in patients who presented with exercise-related joint pain especially in the hip/groin initially attributed to their running. The patients were a 51-year-old female recreational runner, and a 34-year-old male keen road runner. In addition to hip pain, both subjects progressed with pain and stiffness over the second and third metacarpophalangeal joints. The female runner had no symptoms or signs of chronic liver disease or endocrine disturbance. She did, however, have plain radiographs showing degenerative changes in her hands, with hooked osteophytes at second and third metacarpals and both subtalar and talonavicular joints, as well as minor degenerative changes in both hips. The male runner presented with mild hepatomegaly and persistently increased liver function tests, with early fibrosis and heavy iron staining (but no cirrhosis) on liver biopsy. Also, plain x-ray films showed degenerative changes of the hips. In both cases, ferritin concentration was >1000 µg/L. They had a diagnosis of haemochromatosis and were referred to treatment with venesection. Their joint pains and arthropathy continued despite treatment, and they had bilateral total hip replacement. The authors conclude that the diagnosis of haemochromatosis is easily overlooked in patients presenting with exercise-related joint pain if the symptoms are attributed solely to their exercise and sporting activities. Idiopathic haemochromatosis is an inherited disorder of iron metabolism in which excess iron absorption leads to tissue damage associated with characteristic arthropathy. As many as 64% of patients with haemochromatosis develop arthropathy that has been recognized as an early manifestation and predominant clinical factor.
affecting the quality of life of these patients.[26] Phlebotomy, the metal depletion treatment of choice in haemochromatosis, markedly improves survival and prevents complications.[27]

4. Peripheral Arthropathy

With rare exceptions, any joint disorder is capable of presenting initially as monoarthritis. Nonetheless, it is almost always possible to identify patients who need prompt evaluation and treatment to prevent rapid disease progression such as those with suspected septic arthritis. The physician must first attempt to localize the anatomical site of the abnormality. Joint pain may be the result of abnormalities of the joint itself, adjacent bone, surrounding ligaments, tendons, bursae, or soft tissue.[28] The range of disorders causing monoarthritis is listed in table I.

Table I. Differential diagnosis of peripheral arthritis (reproduced from McCune and Golbus,[28] with permission. Copyright © Elsevier 2005)

<table>
<thead>
<tr>
<th>Usually monarticular</th>
<th>Often polyarticular</th>
</tr>
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<tr>
<td><strong>Common</strong></td>
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<tr>
<td>Septic arthritis</td>
<td>Rheumatoid arthritis</td>
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<tr>
<td>bacterial</td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td>tuberculosis</td>
<td>Psoriatic arthritis</td>
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<tr>
<td>fungal</td>
<td>Reactive arthritis</td>
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<tr>
<td>Lyme disease</td>
<td>Calcium pyrophosphate deposition disease</td>
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<tr>
<td>Crystal disease</td>
<td>Most juvenile rheumatoid arthritis and juvenile spondylitis</td>
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<tr>
<td>gout</td>
<td>Systemic lupus erythematosus</td>
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<tr>
<td>pseudogout</td>
<td>Erythema nodosum</td>
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<tr>
<td>Internal derangement</td>
<td>Acute hepatitis B/C</td>
</tr>
<tr>
<td>Haemarthrosis</td>
<td>Rubella</td>
</tr>
<tr>
<td>Trauma or overuse</td>
<td>Lyme disease (usually ≤4 joints)</td>
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<tr>
<td>Pauciarticular juvenile rheumatoid arthritis</td>
<td>Parvovirus</td>
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<tr>
<td>Congenital hip dysplasia</td>
<td>Other crystal-induced arthropathies</td>
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<tr>
<td>Osteochondritis dissecans</td>
<td>Enteropathic arthritis</td>
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<tr>
<td>Haemoglobinopathies</td>
<td>HIV</td>
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<tr>
<td>Loose body</td>
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<td>Paget’s disease involving joint</td>
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<tr>
<td>Stress fracture</td>
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<tr>
<td>Osteomyelitis</td>
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<td>Osteogenic sarcoma</td>
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<td>Metastatic tumour</td>
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<tr>
<td>Synovial osteochondromatosis</td>
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<tr>
<td><strong>Rare</strong></td>
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<tr>
<td>Pigmented villonodular synovitis</td>
<td>Whipple’s disease</td>
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<tr>
<td>Familial Mediterranean fever</td>
<td>Chronic sarcoidosis</td>
</tr>
<tr>
<td>Intermittent hydrarthrosis</td>
<td>Still’s disease</td>
</tr>
<tr>
<td>Behçet’s disease</td>
<td>Pulmonary hypertrophic osteoarthropathy</td>
</tr>
<tr>
<td>Regional migratory osteoporosis</td>
<td>Chondrocalcinosis-like syndromes due to ochronosis,</td>
</tr>
<tr>
<td>Amyloidosis (associated with myeloma or renal failure)</td>
<td>haemochromatosis, Wilson’s disease</td>
</tr>
<tr>
<td></td>
<td>Rheumatic fever</td>
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<tr>
<td></td>
<td>Paraneoplastic syndromes</td>
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<td></td>
<td>Polymyalgia rheumatica</td>
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</table>
Of utmost importance is evaluation for septic arthritis, which is uncommon in the normal joint, but the possibility should be considered in joints recently drained or in patients with arthritis, diabetes mellitus or impaired immune function.[29] Synovial biopsy may play a role in the diagnosis of chronic, unexplained monoarticular arthritis, especially tuberculous synovitis.[28]

Once infection is ruled out, certain rheumatic conditions must always be considered. Common diseases such as RA, gout, and pseudogout, or rarer diseases such as pigmented villonodular synovitis (PVNS) and intermittent hydrarthrosis may present as an acute monoarthritis.

The patient history and physical examination are essential in determining the diagnosis. Inflammatory arthritis is characterized by stiffness of the affected joint that is most noticeable in the morning (morning stiffness of >1 hour) or after a period of inactivity (gelling) and that improves with motion. Also, inflammatory arthritis may often be associated with constitutional symptoms, such as fever or malaise, and involvement of multiple joints.[28]

Occasionally, patients may attend the sports medicine clinic with multiple joint pains (polyarthralgia) or multiple joint pains with synovitis (polyarthritis). In many of these conditions, the diagnosis is clinical. A key diagnostic feature is the onset and pattern of joint involvement along with the extra-articular manifestations. For example, RA typically affects the small joints symmetrically, while reactive arthritis has a propensity to asymmetric involvement of large joints of the lower limb.[29] In table I, we summarize the conditions that present as polyarthritis.

### 4.1 Autoimmune Arthritis

Autoimmune arthritis such as RA and AS have been described to present as a sports-related injury in athletes. Jari and Noble[30] reported five cases of patients presenting with meniscal tears that were subsequently found to have RA. All five patients were middle-aged men who presented with a history and physical signs suggestive of medial meniscal tearing. These signs included a sudden twisting episode, medial joint line tenderness, and a positive McMurray’s sign. Because MRI scans showed effusion and synovial thickening in addition to the medial meniscal tear, full blood count, erythrocyte sedimentation rate, and rheumatoid factor (RF) were also performed. In all patients, the erythrocyte sedimentation rate was raised and the RF was positive. In the three patients who had undergone surgery prior to their RF results being known, the surgical outcome was poor and results of the synovial biopsies were positive for RA. In two patients, surgery was deferred. These patients were successfully treated by a rheumatologist, and surgery was unnecessary. The authors suggest a policy of medical therapy to reduce inflammation in patients clinically suspected or diagnosed with RA before arthroscopic surgery is undertaken.

Juvenile idiopathic arthritis is another diagnosis that should be considered in athletes under the age of 16 years. Juvenile idiopathic arthritis identifies subtypes such as systemic arthritis, oligoarthritis, polyarthritis (RF-positive or -negative), enthesitis-related arthritis, psoriatic arthritis and undifferentiated arthritis. Of special interest is the subtype enthesitis-related arthritis, which mainly affects males over the age of 6 years and is characterized by the association of enthesitis and arthritis. This arthritis commonly affects the joints of the lower extremity, especially the hips. The most common sites of enthesitis are the calcaneal insertions of the Achilles tendon, plantar fascia, and tarsal area. In some cases, arthritis could progress to affect the sacroiliac and spinal joints, thus producing the clinical picture of AS.[31]

Although AS is known and named for spinal involvement, it can initially involve a peripheral joint, especially the shoulders and hips. Hill and Lombardo[32] described a case of AS presenting as shoulder pain in a 22-year-old athlete. This patient denied significant back pain and had been treated earlier with anti-inflammatory agents for the diagnosis of tendonitis. However, upon further questioning, the patient described intermittent episodes of iritis affecting both eyes. Results of his laboratory analyses revealed an elevated erythrocyte sedimentation rate, negative reactions to a RF test and antinuclear antibodies, and a positive test for HLA-B27. A pelvic roentgenogram...
revealed fused sacroiliac joints bilaterally and ischial periostitis with normal hip joints. Roentgenograms of his shoulder showed marked joint-space narrowing, osteoporosis, and joint erosions. Six months after treatment with indomethacin and exercises, the patient had improved range of motion in the shoulder, decreased pain, and he was playing racquet sports with minimal discomfort.

Arthritis is one of the most common manifestations that patients with systemic lupus erythematosus (SLE) exhibit over time. SLE is most often associated with female gender. The symmetric polyarticular pattern is the most common presentation and typically there is no evidence of erosive disease despite a spectrum of inflammatory synovitis and soft tissue swelling around the joints. However, it is usually the associated fatigue that patients complain of as the most debilitating symptom.\(^{[33]}\) Besides arthritis and fatigue, the physicians can confirm diagnosis based on the other systemic signs and symptoms of SLE such as malar or discoid rash, photosensitivity, serositis, haematological, neurological or renal disorders, and positive autoantibodies.

4.2 Crystal-Induced Arthritis

Another aetiology of peripheral arthritis in athletes is gout. Gout is a metabolic disorder characterized by deposition of uric acid crystals in connective tissues and articular cartilage. The onset of gout usually occurs between the ages of 30 and 50 years.\(^{[10]}\) More than 90% of patients with primary gout are men, and the age of peak incidence in men is earlier than that of affected women, who rarely develop the disorder before menopause.\(^{[34]}\)

One case exists of a 33-year-old male marathon runner who was admitted to hospital with a 6-day history of fever, chills, malaise and pain in his right knee and ankle following a 10-mile (16-km) run. A similar episode 2 years earlier had occurred, but instead of knee and ankle, his elbow was affected and was treated with anti-bacterials. He attributed chronic musculoskeletal back and groin pain to running and often took ibuprofen, aspirin (acetylsalicylic acid), or indomethacin for relief. His usual alcohol consumption was about six beers a day. Laboratory data revealed a uric acid level of 10.0 mg/dL (normal 3.4–7.0 mg/dL). The diagnosis was made after aspiration of the knee, which yielded 50 mL of fluid that contained negatively birefringent intracellular crystals. A systolic murmur was also evident on cardiac examination, and an echocardiogram was performed, which revealed mild aortic regurgitation at the site of a nodule on the right coronary leaflet. The authors suggested that this nodule was visceral tophi. The patient was treated with intravenous colchicine and oral indomethacin, and all of his symptoms went into remission. He was discharged on 10 days of oral colchicine followed by allopurinol (300 mg/day) continuously, and 8 months later he was running without difficulty.\(^{[35]}\)

Mair and colleagues\(^{[36]}\) described a case of gout as a source of sesamoid pain in an 18-year-old male intercollegiate wrestler, although gout is extremely rare in teenagers and young adults.\(^{[37]}\) This patient presented with insidious pain in the region of the right first metatarsophalangeal joint and first metatarsal head without a specific prior injury. Radiographs revealed the medial sesamoid to be partitioned with minimal irregularity at the separated margins. It was thought that the patient might have had a stress fracture of the medial sesamoid. He was fitted with a carbon fibre insole and later an extended steel shank, and he refrained from wrestling for 8 weeks. The patient's symptoms resolved, but when he resumed wrestling, he gradually began experiencing more pain. The decision was made to proceed with surgical exploration, which revealed a chalky, white material in the fragments of medial sesamoid that were cystic. Birefringent needles consistent with monosodium urate crystals were identified in the curetted material. The final pathological report confirmed the diagnosis of gout of the medial sesamoid. Following surgery, the patient did well and was able to resume wrestling without pain 3 months later. His serum uric acid level was 7.8 mg/dL (normal 3.5–8.0 mg/dL) and in retrospect, it was noted that his father had longstanding gout of the first metatarsophalangeal joint. It is important to emphasize that
actual determinations of solubility of monosodium urate in human plasma (or serum) indicate that saturation occurs at concentrations of about 7 mg/dL.[37]

Exercise may increase serum uric acid by three mechanisms: (i) adenosine released by exercising muscle may be metabolized by purine nucleoside phosphorylase and xanthine oxidized to produce uric acid; (ii) intense exercise and thermal stress may decrease renal blood flow and clearance of uric acid; and (iii) dehydration decreases plasma volume and increases the concentration of uric acid. Adding the fact that some athletes drink alcohol and take low doses of aspirin for overuse injuries, they are at increased risk for gout.[35]

Pseudogout is a condition associated with deposition of calcium pyrophosphate dehydrate (CPPD) crystals and characterized by joint effusions with marked neutrophilia and a form of secondary osteoarthritis (OA) with a pattern of joint involvement that differs from primary OA. ‘Chondrocalcinosis’ is the term used to define the asymptomatic radiographic finding of calcification of articular or fibrocartilage most frequently related to CPPD deposition.[38] The cause of chondrocalcinosis is unclear, but it has been associated with several medical and hereditary problems. It has been reported to be common in patients with haemochromatosis, primary hyperparathyroidism and gout.[39]

In addition to familial cases, another cause of chondrocalcinosis in young adults is thought to be trauma. Trauma-induced chondrocalcinosis tends to be monoarticular, involving the traumatized joint with degenerative changes, and occurs in relatively young persons without any predisposing medical condition. Trauma-induced monoarticular chondrocalcinosis has been found in internal derangements of the knee, hypermobile joints, and after surgery.[39]

Another case report describes a 20-year-old man with a history of left knee discomfort of several months’ duration while training for and participating in an 800-m run for intercollegiate varsity track. He could not recall an episode of significant trauma and had no history of effusion or locking. He denied any known medical problems, medications, steroid use, or familial history of arthritis. Radiographs revealed what appeared to be a 2- to 3-mm round, calcified, loose body in the lateral compartment of the knee. The differential diagnosis at this time was an osteochondral fracture or osteochondritis dissecans. A knee arthroscopy was performed and revealed thick, white, semisolid material of toothpaste consistency from a cavity at the tibial plateau articular cartilage. This material was excised and under microscope examination was found to be CPPD crystals. Radiographs taken 3 weeks postoperatively revealed that calcification was absent. He remained asymptomatic at 10 months’ follow-up and resumed his normal schedule. The authors suggested that chondrocalcinosis must be included in the differential diagnosis of intra-articular calcified lesions in young athletes with knee pain.[39]

4.3 Lyme Disease

Sportsmen, backpackers, and outdoor athletes may acquire unusual infectious diseases in the fields and forests of the US. One of these conditions, Lyme disease, is a tick-borne multisystem disorder caused by the spirochete Borrelia burgdorferi. It characteristically initiates with a particlar lesion on the skin known as erythema migrans, which may be followed by neurological, cardiac, or articular abnormalities. Inflammatory arthritis is the most recognized feature of persistent infection, and approximately 60% of untreated patients develop monoarticular or asymmetric oligoarticular arthritis primarily in the large joints, with objective physical findings of synovial thickening or joint effusion. The diagnosis of Lyme disease is a clinical one. The serological test should be used to confirm the clinical diagnosis.[40,41]

Seldes and colleagues[41] have described a 20-year-old male university football player who presented with an atraumatic spontaneous haemarthrosis of the left knee. This patient denied recent infection, fever, chills, night sweats, history of bleeding abnormalities, or sexually transmitted diseases. Eight months before his clinic visit, the patient noticed a ‘red splotchy rash’, which was diagnosed as an allergic reaction by a
dermatologist. Laboratory examinations, radiographs, and MRIs were not helpful. The diagnosis of Lyme arthritis was made based on his clinical examination and an unequivocally positive serological testing. After the treatment, which consisted of intravenous ceftriaxone 1 g/day for 14 days, the patient was asymptomatic.

4.4 Pigmented Villonodular Synovitis (PVNS)

Another uncommon cause of monoarthritis is PVNS. This benign disorder results in an increased proliferation of synovium, causing villous or nodular changes of synovial-lined joints, bursae, and tendon sheaths. Most patients present with monoarticular swelling, haemarthrosis, and a gradual increase in pain. The knee is the most common location followed by the hip, ankle and shoulder. Radiographic evaluation of PVNS often depicts increased soft-tissue density and may show bony erosions. MRI reveals low-signal intensity on both T1- and T2-weighted images. Histological examination confirms the diagnosis of PVNS. Partial or complete synovectomies are considered the treatment of choice.[42,43]

A 30-year-old sportsman presented with knee pain after several twisting episodes during a game of football. On examination, there was specific anteromedial joint line pain, and results of a McMurray’s test were positive. A diagnosis of a medial meniscal tear was made. Arthroscopy, however, revealed a large pedicular lesion originating from the insertion of the anterior horn of the medial meniscus. Mini-arthrotomy was performed, and the diagnosis of PVNS was made after histological examination.[42]

Mitchell and colleagues[44] reported a case of an 18-year-old division III college football player with a previous history of chondromalacia patella, who sustained a twisting injury of the right knee 3 days before presentation. He had a negative Lachman’s test. As he had a moderate effusion, aspiration of the joint was performed and yielded serosanguineous fluid. The clinical diagnosis was chondromalacia patella with irritation. After 6 months he returned complaining of a 1-month history of swelling and tenderness in the peripatellar area and over the vastus medialis muscle. On physical examination, he had a moderate effusion and crepitus over the lateral aspect of the patella and a possible intra-articular mass in that region. The clinical diagnosis at that time include a possible loose body with chondromalacia patella and a possible medial meniscal tear. Besides chondromalacia patella, MRI showed diffuse synovitis with probable PVNS. Arthroscopy was performed with shaving chondroplasty of the patella, and subtotal synovectomy. Histological evaluation confirmed the diagnosis of PVNS.

Saxena and Perez[43] published a review of ten athletic patients with PVNS about the ankle. Most of the patients were previously involved in lateral motion sports such as basketball, tennis, soccer, or aerobics. Two patients ran for exercise. Nine of ten patients had a history of ankle sprains, with pain laterally. Plain radiographs showed bony changes about the talus and adjacent bones in four of ten patients; MRI showed PVNS findings in all ten. PVNS was found in multiple sites about the ankle including three ankle joints and two subtalar joints. All patients had synovectomy and tenosynovectomy, and eight were able to return to sports participation 4–12 months after surgery.

5. Soft Tissue Disorders

Musculoskeletal soft tissue injuries are a leading cause of pain and disability in both competitive and recreational athletes. Incidence estimates are as high as 50% in distance runners.[45] Many of these injuries are acute tears or strain or are provoked by chronic muscle-tendon overload or overuse and muscle fibre ‘microtrauma’. However, these disorders may occasionally be a manifestation of a systemic rheumatic disease.

5.1 Bursitis

Bursae are small fluid-filled sacs located between tendons, muscles and bones that serve to cushion and reduce friction. Bursitis is defined as inflammation of these superficial or deep bursal sacs. The most common sites of bursitis included the olecranon bursa over the elbow region, the
trochanteric bursa in the hip, and the overlooked anserine bursa over the medial aspect of the knee. The presenting symptom is pain. Obvious swelling is seldom present except in the most superficial areas such as the elbow or knee. Besides local irritation and infection, bursitis can be associated with gout. Olecranon bursitis is especially more common in patients with gout or RA. In these cases, aspiration can be useful to determine the aetiology of the disorder.\[46\]

Septic bursitis should always be considered for patients who have diabetes or who use intravenous drugs as well as for anyone who is immunocompromised. Patients with infected bursae usually have exquisite tenderness, redness, and heat over the bursal site and may have an elevated body temperature.\[46\]

5.2 Tendinopathies

Tendinopathies are often related to increased age, muscle imbalance, and anatomic malalignment. The most common sites are the supraspinatus, finger flexors, patellar and Achilles tendons.\[46\]

Some tendinopathies, particularly lateral epicondylitis, have been associated with certain sports and occupational activities, in keeping with a presumed mechanical aetiology. One study found an increased prevalence of RF in patients with repeating lateral epicondylitis and wrist tenosynovitis suggesting that there may be an underlying predisposition to generalized rheumatic disease in some cases.\[47\]

The frequency of tendon involvement in patients with RA has been reported to be as high as 64%, although most studies report lower frequencies. The most frequent sites for rheumatoid tendonitis are the hand extensors and flexors.\[48\] This is typically seen in the setting of synovitis and/or deformity.

The number and incidence of tendon injuries in general have increased substantially during the last few decades, and this increase has been dominated by problems with the Achilles tendon. Because most Achilles tendon injuries take place in sports and there has been a common surge in sporting activities, the number and incidence of the Achilles tendon overuse injuries and complete ruptures have increased in industrialized countries. In general, Achilles tendon problems arise from two origins: (i) excessive loading-induced degeneration of the tendon; and (ii) the tendon becomes the site for a systemic disease (e.g. gout, pseudogout, spondyloarthropathies and RA).\[48\]

Although there is no description of tendon rupture in athletes due to rheumatic disease, it is described that RA, gout, CPPD deposition and SLE can lead to tendon rupture in young individuals. Among all tendon ruptures in RA, the most frequently ruptured tendons are hand extensors; all the other locations are clearly less common.\[48\] In RA, tendon rupture may occur, possibly by the overproduction of matrix metalloproteinases.\[49\] Gout has been described as a cause of peroneus tendon rupture in the presence of tophaceous gouty infiltration.\[50\] Although rare, extensor tendon rupture at the wrist associated with CPPD deposition has been described.\[51\] SLE can cause nonerosive joint deformities, but rarely can lead to spontaneous tendon rupture.\[52\]

5.3 Enthesitis

Enthesitis is defined as any pathological condition involving the entheses. The entheses are any point of attachment of skeletal muscles or ligaments to bone, where recurring stress or inflammatory autoimmune disease can cause inflammation or occasionally fibrosis and calcification. Plantar fasciitis is considered an enthesitis, and it is a common diagnosis in athletes usually having a mechanical aetiology. Other common sites are the ischial tuberosities, greater trochanters, spinous processes, costochondral and manubriosternal junctions, and iliac crests.\[8\]

Enthesitis is also one of the main diagnostic criteria of spondyloarthritis, especially AS. However, spondyloarthritis presents with other inflammatory features such as low back pain with stiffness, peripheral asymmetric arthritis and elevated acute-phase reactants.\[6\]

5.4 Carpal Tunnel Syndrome

Carpal tunnel syndrome (CTS) is the most commonly diagnosed compression neuropathy in Rheumatic Diseases and Sports-Related Injuries 927
the upper extremity. It usually begins as an isolated phenomenon, but symptoms of CTS can accompany many systemic diseases, such as congestive heart failure, amyloidosis associated with multiple myeloma, hypothyroidism and tuberculosis. More commonly, CTS is associated with conditions such as pregnancy, diabetes, obesity, and any inflammatory arthritis affecting the wrist such as RA and gout.[53] The classic constellation of symptoms consists of weakness or clumsiness of the hand; paraesthesias in the thumb, index, and long fingers; and nocturnal paraesthesias in the affected digits. Patients may often complain of forearm and elbow pain that is aggravated by activities, but is poorly localized and aching in nature. The diagnosis of CTS is usually clinical. Bilateral electrodiagnostic tests should be used to confirm the diagnosis, particularly in patients with significant motor loss, atrophy or constant sensory loss or in patients with atypical signs or symptoms.[53]

6. Practical Recommendations

Effective management of athletes presenting with musculoskeletal complaints requires a structured history, physical examination, and definitive diagnosis that distinguish the soft tissue problem from a joint problem and an inflammatory syndrome from a noninflammatory syndrome. Clues to a systemic inflammatory aetiology may include constitutional symptoms, morning stiffness, elevated acute-phase reactants and progressive

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CCP = cyclic citrullinated peptide; CPPD = calcium pyrophosphate dehydrate; CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; HLA = human leucocyte antigen; MCP = metacarpophalangeal; MRI = magnetic resonance imaging; MTP = metatarsophalangeal; PIP = proximal interphalangeal.
symptoms despite modification of physical activity. Also, the lack of injury mechanism suggests an underlying disease. In these circumstances, more complete workup is reasonable including radiographs, MRI and laboratory testing for autoantibodies. Table II summarizes the main clinical features and laboratory tests in the most common rheumatic diseases.

7. Conclusion

There are many case reports of rheumatic diseases masquerading as a sports medicine condition. In the athlete with complaints of swollen joints, low back pain with stiffness, systemic symptoms, and without a history of trauma, inflammatory causes should be considered. The astute sports medicine practitioner should maintain an index of suspicion for rheumatic diseases as adequate and prompt treatment can modify disease progression, allowing the athlete to continue with regular exercise programmes.

Acknowledgements

No sources of funding were used in the preparation of this review and the authors have no conflicts of interest directly relevant to its contents.

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