Clinical

The acutely swollen knee
Part 1: Management of atraumatic pathology

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Abstract

The acutely swollen knee is a common presentation of knee pathology in the Emergency Department and the primary care setting whether on board ship, a Regimental Aid Post or Medical Centre. The swollen knee has both traumatic and atraumatic (systemic) causes, all of which can be accurately diagnosed with an understanding of the underlying injury patterns and patho-anatomy. In Part One, we will be examining the management of non-traumatic causes, followed by Part Two, looking at traumatic causes, in the next issue of the Journal. A detailed clinical history combined with thorough clinical examination will establish the diagnosis, or at least the narrow differential diagnosis in the majority of cases. The uses of specialist examination techniques, diagnostic imaging and arthrocentesis can further assist the clinician in confirming the correct diagnosis and thus prescribing the appropriate treatment.

This review will endeavour to give a consensus of opinion and structured guidelines in the diagnosis and initial management of patients presenting with acute or recent-onset swelling of the knee related to atraumatic pathology.

Introduction

A swollen knee or knee effusion is a common problem caused by the accumulation of fluid in or around the knee joint. Knee effusions may due to traumatic or atraumatic causes. The most common traumatic causes are fracture or ligamentous and meniscal injuries, and will be discussed in Part Two of this article in the next issue. Atraumatic causes include arthritis, infection, crystal deposition, haematogenous and those resulting from tumours. Effusions can have different rates of onset due to different causes. Rapid swelling after injury usually indicates bleeding into the joint or haemarthrosis caused by a damaged and bleeding intrarticular structure, whereas rapid swelling without injury could indicate infection or crystal arthropathy. Delayed swelling with trauma could indicate meniscal injury or ligamentous sprain; an absence of trauma could indicate arthritis. A detailed history combined with thorough clinical examination will establish the diagnosis or at least a narrow differential diagnosis in the majority of cases. The use of specialist examination techniques, diagnostic imaging and arthrocentesis can further assist the clinician in confirming the correct diagnosis and thus prescribing the correct treatment that the patient requires.

The aim of this paper is to perform a literature review so as to gain a consensus of opinion and structured guidelines in the diagnosis and initial management of patients presenting with acute or recent onset swelling of the knee relating to atraumatic pathology.

Definition

A recent collaboration between a committee comprising orthopaedic surgeons and rheumatologists from the European Federation of National Associations of Orthopaedics and Traumatology (EFORT) and The European League Against Rheumatism (EULAR) published a combined consensus definition of the acutely swollen knee which will be used in this paper (1). They concluded that the definition should be: all patients newly presenting to a doctor with a history or examination finding suggesting onset of swelling (defined as an increase in volume) within a recent time (up to four to six weeks) in any anatomical structure of the knee, intra-articularly and peri-articularly. The term “acute” in isolation for this review was felt to suggest an emergency and that the patient would need to be reviewed without delay. It was also deemed appropriate to include the term “recent onset swelling” in order to be more encompassing of all types of knee effusion i.e. up to four to six weeks. An example of the appearance of an acutely swollen knee is shown in Figure 1.
Epidemiology
Robust and well-structured epidemiological studies have not been conducted into the incidence and prevalence of the symptom of acute or recent onset of swelling of the knee. However, the lifetime prevalence of swelling of the knee has been reported as 27% in a British study (2) and 10% in a European study (3). Lifetime prevalence of knee symptoms is 54% in the same British study and 17% in a Scandinavian study (4).

MANAGEMENT OF SPECIFIC ATRAUMATIC CAUSES
i. INTRODUCTION
Delayed or inadequate treatment of septic or infectious arthritis can lead to irreversible joint damage and disability with a significant mortality rate of 11% (5). It is, therefore, imperative that rapid assessment and subsequent diagnosis occurs to enable prompt treatment.

Classic teaching states that patients usually have an acute onset of arthralgia and swelling over less than two weeks. Physical examination can reveal fever, localised erythema and a reduced range of movement, combined with reluctance to weight-bear and a hot, swollen joint. There are well-recognised risk factors: a previous abnormal joint, immune-compromised status, intravenous drug use, rheumatoid arthritis (RA) or osteoarthritis (OA), diabetes, previous intra-articular corticosteroid injections and cutaneous ulcers. The most important investigation is drawing synovial fluid from the joint (arthrocentesis) and examination of the fluid. Organisms present on Gram stain and positive cultures can confirm the diagnosis. An elevated peripheral white blood cell count (WCC), C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) are also indicative of a potential infectious process. Blood cultures can also aid diagnosis and organism identification.

A recent systematic review has demonstrated that the overall impression of a clinician experienced in assessing joint disease is the only gold standard for diagnosing septic arthritis. The review stated that this had proven superior to any laboratory or radiological investigation (6). It concluded that if septic arthritis is suspected, “then advice from a musculoskeletal specialist should be sought at the earliest opportunity”. This is an important guiding principle that must be considered by all health professionals likely to encounter patients presenting with an acutely swollen knee.

Presentation
60% of all cases reported involve the hip and knee (7,8), but in up to 22% of cases more than one joint is affected, indicating that a poly-articular presentation does not exclude the diagnosis of septic arthritis (9). In addition, many studies have demonstrated that fever is not a reliable indicator of a septic joint (5,9). Furthermore, if there is a pre-existing inflammatory joint disease present, then pain out of proportion to the patient’s usual pain, or the pain usually felt in other joints, should increase concern about the diagnosis of an infected joint.

Investigations
Numerous studies have shown that the presence of a normal WCC, ESR or CRP levels does not exclude the diagnosis of sepsis (5). However, the use of these three tests may be useful in monitoring the response to treatment.

Synovial fluid culture has been shown to be more sensitive than microscopy alone, as synovial fluid Gram staining is only positive in 50% of cases (10). Hindle et al (11) demonstrated that detection of an organism on initial microscopy or on subsequent culture of their aspirate is less reliable if the patient had received antibiotics. The sensitivity of microscopy and culture dropped from 46% to 0% and from 69% to 21% respectively, indicating the importance of educating all clinicians to withhold antibiotics until microbiology samples have been taken e.g. by joint fluid aspiration. This concern is obviously overridden when the patient is clinically unstable and systemically septic or in a case of prolonged transport time to an orthopaedic specialist. In this situation gaining physiological stability is of greater importance than microbiology specimens. In addition, the importance of blood cultures should never be underestimated as they may identify the causative organism when synovial fluid culture fails (10). It has been shown that synovial fluid WCC counts are not sufficiently reliable to exclude or confirm a diagnosis of septic arthritis (12).

Microbiology
The Staphylococci and Streptococci account for 91% of cases of septic arthritis (5, 9,10). Older and immune-compromised patients usually suffer from gram-negative sepsis, whereas anaerobes are more commonly seen in penetrating trauma.

Imaging
Radiographs are usually taken to rule out other pathology, including osteomyelitis. Magnetic resonance imaging
(MRI) is the only imaging modality that can apparently reliably demonstrate the presence of infection, but in these studies no controls were used and this finding needs to be confirmed with further research (10).

**Treatment**

We found no studies that could statistically prove a difference between an arthroscopic or open washout and debridement of a septic joint. The overriding conclusion though is that this should be performed in a timely manner to prevent the sequelae of joint destruction and mortality previously reported. The management of septic arthritis is summarized in Box 1.

**Box 1. Management of septic arthritis**

<table>
<thead>
<tr>
<th>Pre-shore / pre-hospital management</th>
</tr>
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<tbody>
<tr>
<td><strong>History</strong></td>
</tr>
<tr>
<td>Acute onset knee swelling with no history of trauma (&lt;2weeks). Any associated risk factors: previous abnormal joint, immune-compromised status, intravenous drug use, diabetes, previous intra-articular corticosteroid injections or cutaneous ulcers. Polyarticular presentation does not rule out the diagnosis of septic arthritis.</td>
</tr>
<tr>
<td><strong>Examination</strong></td>
</tr>
<tr>
<td>Pyrexia, localized erythema, joint warmth, a reduced range of movement combined with reluctance to weight bear but with stable ligaments.</td>
</tr>
<tr>
<td><strong>Radiographs</strong></td>
</tr>
<tr>
<td>(if available) Essentially normal.</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
</tr>
<tr>
<td>If a septic joint is suspected then it is imperative to seek advice from a musculoskeletal specialist clinician at the earliest opportunity. This clinician should be skilled in the technique of arthrocentesis and have a laboratory available to give immediate microscopy and blood test results. Only in the very rare event that either patient transfer is limited by military constraints, or that the patient is in extreme systemic sepsis, should antibiotics be given before arthrocentesis and review by a musculoskeletal specialist.</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Hospital management</th>
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<tbody>
<tr>
<td><strong>History, Examination and Radiographs as above</strong></td>
</tr>
<tr>
<td>Blood FBC, CRP, ESR, Urea and electrolytes, cultures. Arthrocentesis: Urgent microscopy and then culture. Organisms seen on gram stain, WBC &gt; 50 000/mm3 or positive cultures are all suggestive of septic arthritis.</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
</tr>
<tr>
<td>If a septic joint is suspected then it is imperative to seek advice from a musculoskeletal specialist clinician at the earliest opportunity. Open or arthroscopic debridement of the joint should occur in a timely manner to prevent the sequelae of joint destruction.</td>
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**ii. CRYSTAL ARTHROPATHY**

**A. GOUT**

Gout arises from the deposition of urate crystals in joints, with an associated inflammatory response. Crystal deposition occurs when serum becomes saturated with urate, the end product of the purine metabolism. The prevalence of gout is at least 1% in the UK. Gout is much more common in men than in women (20:1) and in the older population (>55) (13).

**Presentation**

The most common presentation of gout is an acute mono-arthritis of the great toe (podagra). The onset of symptoms is usually rapid. Gout can affect any joint, but the foot, ankle, knee, wrist, finger, and elbow are the usual sites if the great toe is not involved. Crystal deposits (tophi) may also develop around these joints but most commonly the hands, feet, elbows, and ears. An acute attack usually lasts for just over a week. Some patients only have one or two attacks over a lifetime with no sequelae, whilst others may have frequent attacks and be left with permanent joint damage. Subcortical cysts without erosions, peri-articular swellings and chondrocalcinosis are the characteristic signs seen on radiographic examination.

**Diagnosis**

The gold standard diagnostic confirmation of gout is by synovial fluid microscopy using polarised light, with which method urate crystals can be identified as strongly negatively birefringent, needle-shaped intracellular crystals. However, the utility of this investigation can be limited, as reports state that this is only demonstrated in 11% of cases. This could be due to a number of reasons. Firstly, obtaining fluid can be difficult, especially from the first metatarsophalangeal joint, or if the clinician is inexperienced in arthrocentesis. Secondly, discarding the needle used for aspiration or a delay in microscopic examination can lead to failure despite a potentially diagnostic aspirate. Finally, access to a laboratory that provides this test and accurate identification of these crystals has varied greatly across the country in the past (14). Consequently, the American College of Rheumatology requires six or more of the criteria listed below to be present in order to make a clinical diagnosis (Box 2). The management of gout is summarized in Box 3.
Investigation
Arthrocentesis: To identify urate crystals and exclude infection. Bloods: FBC, Urea and electrolytes and CRP to help rule out septic arthritis. Serum Uric Acid (SUA) levels are usually within normal limits during an acute attack but a base line level should be recorded. Radiograph: Subcortical cysts without erosions, peri-articular swellings and chondrocalcinosis are the characteristic signs.

Treatment
The British Society for Rheumatology recently produced guidelines for the treatment of gout (Box 4). This focused on three main areas of treatment: management of acute gout, recommendations for diet and lifestyle modifications, and management of recurrent and chronic gout (15).

Pre-shore / pre-hospital management
History
Acute onset knee swelling with no history of trauma (in last two weeks). Any associated risk factors: previous gout attack, diuretic use, known high SUA and high purine diet. Poly-articular presentation does not rule out the diagnosis of gout.

Examination
Pyrexia, localized erythema, joint warmth, a reduced range of movement combined with reluctance to weight bear but with stable ligaments.

Radiographs
(if available) Subcortical cysts without erosions, peri-articular swellings and chondrocalcinosis.

Hospital management
History, Examination and Radiographs as above
Investigations: Blood
FBC, CRP, ESR, Urea and electrolytes, cultures to exclude septic arthritis.

Arthrocentesis
Urgent microscopy and then culture. Strongly negatively birefringent needle shaped intracellular crystals should be seen using polarized light microscopy.

Treatment
Open or arthroscopic debridement of the joint is sometimes required if there is any concern that septic arthritis is coexistent. This should occur in a timely manner if suspected and can aid in both symptom control with additional microbiology sampling. However it is imperative that the advice from a musculoskeletal specialist clinician should be sought at the earliest opportunity to make this decision. Acute and chronic treatment by modification of diet and lifestyle should follow.
Box 4. British Society for Rheumatology Guidelines For the Management of Gout

<table>
<thead>
<tr>
<th>Exclude Septic Arthritis &amp; suppress pain and inflammation</th>
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<tbody>
<tr>
<td>NSAID +/- PPI or Colchicine or Corticosteroid (i.a,oral,i.m,i.v.)</td>
</tr>
<tr>
<td>Resolution</td>
</tr>
</tbody>
</table>
| Further attacks (or risk factors+++)
  Treat acute attack: when resolved add
  Allopurinol + prophylactic cover with low dose NSAID +/- PPI or Colchicine
  Titrate allopurinol dose: depending on SUA may require doses up to 900mg/day
  DO NOT STOP ALLOPURINOL DURING ACUTE ATTACKS |
| No Renal Impairment
  Change to Sulphinpyrazone or Benzbromarone or Probenecid |
| Renal Impairment
  Change to Benzbromarone Consider combination therapy with low dose allopurinol |

i. Acute Gout
Affected joints should be rested, with non-steroidal anti-inflammatory drugs (NSAIDs) being commenced immediately (if no contraindications) and continued for one to two weeks. In patients with an increased risk of peptic ulcers, bleeds or perforation, co-prescription of ulcer-preventing agents is advised. Oral corticosteroids are an effective alternative in patients unable to tolerate NSAIDs. A recent randomised controlled trial (RCT) described equally effective resolution of symptoms when using either naproxen or oral prednisolone (16). Colchicine is another alternative therapy, but leads to common side effects of diarrhoea and nausea that are not well tolerated by patients. If diuretic drugs are being used to treat hypertension, an alternative antihypertensive agent should be considered. Allopurinol should not be prescribed during an acute attack, but if allopurinol is already being taken then it should be continued and the acute attack should be treated as described above.

ii. Recommendations for diet and lifestyle modifications
Overweight patients should have dietary advice in order to achieve an ideal body weight. This should be attempted in a controlled fashion with the intake of protein and high purine content foods being restricted (red meat, liver, kidneys, shellfish etc). Alcohol consumption should be restricted to less than the weekly-recommended amount. Men should drink no more than 21 units of alcohol per week, no more than four units in any one day, and have at least two alcohol-free days a week. Women should drink no more than 14 units of alcohol per week, no more than three units in any one day, and have at least two alcohol-free days a week.

iii. Management of recurrent and chronic gout
The SUA should be maintained below 300 umol/l by using uric-acid-lowering drug therapy. This should be started if a second attack occurs within a year. Uric-acid-
lowering treatment should also be offered to patients with renal insufficiency and to those who need to continue treatment with diuretics. This should be delayed until one to two weeks after the acute symptoms have resolved. Colchicine 0.5mg b.d. should be co-prescribed on initiation of treatment with allopurinol and continued for up to six months (side-effect dependent). NSAIDs can be used as an alternative, if not contraindicated, but this should be limited to six weeks only. This adjunctive therapy has been proven to prevent early re-occurrence. In patients who fail to respond to allopurinol, alternative or adjunctive drugs can be used; sulphinpyrazone (200-800 mg/day) or benzbromarone (50-200 mg/day) in patients with mild/moderate renal insufficiency. Cardiovascular events have an increased prevalence in gout sufferers and so the primary care provider should implement routine monitoring. Regular blood pressure, cholesterol and diabetes tests are recommended.

**B. PSEUDOGOUT**

Pseudogout can be defined as acute attacks of synovitis induced by calcium pyrophosphate dihydrate (CPPD) crystals, which clinically resemble gouty arthritis due to monosodium urate (MSU) crystal deposition. It mainly affects older people, with over 50% of all octogenarians displaying some evidence of articular chondrocalcinosis (17). There are no published reports of prevalence in younger people and there is no major gender predominance. However, acute attacks occur more commonly in men (18).

**Presentation**

Pseudogout attacks may take longer to reach peak intensity and subsequent recovery compared to gout, despite adequate treatment. The knee is the most commonly involved joint, followed by the wrist, ankle, elbow, toe, shoulder and hip. Localised erythema, joint warmth, reduced range of movement, combined with reluctance to weight-bear, are the common presenting symptoms. Patients are usually asymptomatic between attacks. HAemochromatosis and chronic hypomagnesaemia due to Gitelman’s and Bartter’s syndromes are known to be strongly associated with pseudogout.

**Diagnosis**

The gold standard diagnostic confirmation of pseudogout is by synovial fluid microscopy using polarized light. CPPD crystals can be identified as rhomboid-shaped, rod-like crystals that exhibit weakly positive or no birefringence by this method.

**Investigation**

Arthrocentesis: Exclude sepsis. Confirm CPPD crystals as described above. Bloods: FBC, Urea and electrolytes and CRP to help rule out septic arthritis. Ferritin, iron saturation, transferrin, calcium and magnesium should be assessed due to the associations mentioned above.

Radiograph: Chondrocalcinosis is the characteristic sign seen on radiographic examination of the knee.

**Treatment**

Affected joints should be rested with NSAIDs being commenced immediately (if no contraindications) and continued until symptoms settle. In patients with increased risk of peptic ulcers, bleeds or perforation, co-prescription of ulcer preventing agents is advised. Oral corticosteroids and colchicine are an effective alternative in patients unable to tolerate NSAIDs. Methotrexate has been shown to decrease the frequency and intensity of recurrent attacks of pseudogout (19).

**iii. OSTEOARTHRITIS**

Osteoarthritis (OA) is a common chronic and progressive musculoskeletal disorder that involves all tissue components of the joint: bone, cartilage, synovium, muscle and ligaments. It is characterised by progressive loss of articular cartilage (softening, fibrillation and then ulceration), leading to sclerosis and eburnation of subchondral bone. Subchondral cyst and osteophyte formation are later features (20).

**Presentation**

OA is the most common form of arthritis in the UK, with more than 6 million people having painful osteoarthritis in one or both knees. Prevalence increases with age; with one in five adults aged 50-59, and almost half of all adults aged over 80 having painful osteoarthritis in one or both knees (21). Common symptoms of patients with OA include gradual onset of swelling with loss of function. The pain is usually worse with function and better with rest. The joint is not usually extremely warm and the majority of patients are of an age greater than 50 years (21).

**Diagnosis**

The presentation and radiographic findings are usually sufficient to be able to make the diagnosis of OA. However, MRI and ultrasound are also validated non-invasive methods of assessing OA (22). Radiographic findings include joint space narrowing and sub-chondral sclerosis followed by sub-chondral cyst and osteophyte formation. In the knee this should be assessed by weight bearing radiographs in two planes. Septic arthritis, as well as other differentials, should always be excluded before treatment is commenced using the guidelines above.

**Treatment**

Non-Operative

Non-operative treatments should always be exhausted before any surgical options are considered.

i. Muscle-strengthening physiotherapy regimes have been shown to be beneficial if there is a high degree of patient compliance (23,24).

ii. Weight loss has obvious general health benefits, but
evidence indicating that this halts the progression of OA is variable (25).

iii. Bracing has been shown to be successful in managing the symptoms of OA but is poorly tolerated by patients mainly due to skin complications (26).

iv. NSAIDs and Paracetamol have been shown to be very effective in pain management (27). However, the long-term side effects of NSAIDS pose limitations in this regard.

v. Recent Cochrane reviews demonstrated that intra-articular steroids are beneficial in the management of OA, but have only a short-term effect (28).

vi. Further Cochrane reviews indicate that Hyaluronan (HA) viscosupplementations of the knee were not cost effective compared to other OA treatments. However, they were found to be beneficial, and comparable to other systemic treatments (29).

Operative
Surgical treatment of OA is beyond the scope of this review, but can range from osteotomy to partial or total knee replacement.

The management of OA is summarized in Box 5.

Box 5. Management of osteoarthritis

| Pre-shore / pre-hospital management |
| History                          |
| Gradual onset pain and swelling with loss of function. Pain is usually worse with function and better with rest. Poly-articular presentation common. |

| Examination                      |
| Apyrexial, no systemic symptoms, reduced range of movement combined with an antalgic gait. Crepitus and deformity are seen in more advanced disease. |

| Radiographs                     |
| (if available) Joint space narrowing and subchondral sclerosis with subchondral cyst and osteophyte formation in more advanced disease. |

| Treatment                       |
| Non-operative measures as listed above, or orthopaedic specialist referral to decide on surgical management, which may range from arthroscopic microfracture and biologic treatments to osteotomy and/or partial or total knee replacement. |

| Hospital management             |
| History, Examination and Radiographs as above |
| Investigations: Bloods to rule out other pathologies. |
| Treatment                       |
| Orthopaedic specialist referral to decide on surgical management, which may range from osteotomy to partial or total knee replacement. |

iv. RHEUMATOID ARTHRITIS
Rheumatoid Arthritis (RA) is a chronic, progressive autoimmune disease causing soft tissue swelling and inflammation of the lining of the joints. Bone erosions and osteopenia result in painful deformity and immobility.

Presentation
RA is the second most common form of arthritis in the UK and the most common inflammatory joint disorder. The prevalence in the UK has been reported as 2% of all adult women and less than 0.5% of men (around 400,000 adults in 2006) (30). The small joints of the hand or feet are usually the first to be affected. Symptoms vary depending on the severity of the disease process.

Box 6. Management of rheumatoid arthritis

| Pre-hospital and hospital management |
| History                          |
| Gradual onset pain and swelling with progressive deformity and loss of function. Swelling often presents with acute exacerbations. Poly-articular presentation is common especially of the small joints of the hands and feet. |

| Examination                      |
| Swelling, warmth, synovial thickening, tenderness, reduced range of movement combined with an antalgic gait. Crepitus, severe deformity and systemic manifestations can be seen in more advanced disease. |

| Investigations                   |
| FBC, urea and electrolytes and CRP to help rule out septic arthritis. Rheumatoid factor (RhF) and ESR can also aid diagnosis. However RhF can be found in people without RA or with other autoimmune disorders. In general, though, if rheumatoid factor is not present in someone with RA, then the course of the disease is less severe. Arthrocentesis can reveal elevated synovial fluid protein levels, decreased glucose levels and synovial fluid WBC count ranging from 2,000 to 50,000 per mm3 (2 to 50 x 109 per L). This is suggestive of any inflammatory process, not only RA. |

| Radiographs                     |
| Joint space narrowing, peri-articular osteopenia, marginal erosions, and peri-articular soft tissue swellings. |

| Treatment                       |
| Referral to a rheumatology specialist. Pharmacological treatments range from simple analgesia to disease-modifying anti-rheumatic drugs (DMARDs) such as methotrexate and TNF-alpha inhibitors e.g. etanercept. If non-operative measures fail to control symptoms then orthopaedic interventions (ranging from arthroscopic/open synovectomy to arthroplasty) can be used. |
Diagnosis

The management of RA is summarised in Box 6. The diagnosis of RA is complex, should be made by a specialist rheumatologist and is beyond the scope of this article. However, as a point of reference, the American College of Rheumatologists published diagnostic clinical criteria guidelines in 1987 to aid in this process (31), as described in Box 7.

v. HAEMARTHROSIS

Bleeding into a joint is referred to as haemarthrosis and is an important cause of mono-articular joint pain and swelling. Rapid, large swelling (within a few hours) in association with trauma is indicative of a haemarthrosis due to fracture or significant intra-articular injury. A haemarthrosis without associated trauma may well be the result of haemophilia (or other bleeding disorder), neoplasm, OA, pigmented villonodular synovitis or oral anticoagulant therapy. Arthrocentesis can be performed to aid in diagnosis and to alleviate pain. However this is contraindicated if a diagnosis of neoplasm or bleeding disorders is suspected. The use of arthrocentesis in patients on anticoagulant therapy is not advised due to the high rates of recurrence and the risk of introducing infection. Haemophiliacs and patients with other bleeding disorders should be assessed and treated in a specialist unit, which is beyond the scope of this review.

vi. TUMOUR

Primary bone tumours most commonly present around the knee, but only represent 0.2% of all tumours (32). Both benign and malignant tumours can present with a knee effusion. Symptoms such as fever, night sweats, unintentional weight loss and night pain should alert the clinician to the possibility of a neoplastic process. Plain radiographs can usually rule out a bone lesion. This diagnosis should always be ruled out of the differential diagnosis before embarking on arthrocentesis or other invasive investigations. These patients should be reviewed by an orthopaedic specialist who will subsequently refer on to a Specialist Bone Tumour Unit, of which there are only a few in the UK.

Baker’s cyst

A Baker’s cyst is a benign swelling found in the popliteal fossa. The cyst arises from the synovial sac of the knee joint, and is usually associated with a form of knee arthritis (OA or RA), or occasionally with a meniscal tear. Baker’s cysts

**Box 7. American college of Rheumatology Rheumatoid Arthritis Classification 1987**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Morning stiffness</td>
<td>Morning stiffness in and around the joints, lasting at least 1 hour before maximal improvement</td>
</tr>
<tr>
<td>2. Arthritis of three or more joint areas</td>
<td>At least three joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician. The 14 possible areas are right or left PIP, MCP, wrist, elbow, knee, ankle, and MTP joints</td>
</tr>
<tr>
<td>3. Arthritis of hand joints</td>
<td>At least one area swollen (as defined above) in a wrist, MCP, or PIP joint</td>
</tr>
<tr>
<td>4. Symmetric arthritis</td>
<td>Simultaneous involvement of the same joint areas (as defined in 2) on both sides of the body (bilateral involvement of PIPs, MCPs, or MTPs is acceptable without absolute symmetry)</td>
</tr>
<tr>
<td>5. Rheumatoid nodules</td>
<td>Subcutaneous nodules, over bony prominences, or extensor surfaces, or in juxta-articular regions, observed by a physician</td>
</tr>
<tr>
<td>6. Serum rheumatoid factor</td>
<td>Demonstration of abnormal amounts of serum rheumatoid factor by any method for which the result has been positive in ≤5% of normal control subjects</td>
</tr>
<tr>
<td>7. Radiographic changes</td>
<td>Radiographic changes typical of rheumatoid arthritis on postero-anterior hand and wrist radiographs, which must include erosions or unequivocal bony decalcification localized in or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify)</td>
</tr>
</tbody>
</table>

For classification purposes, a patient shall be said to have rheumatoid arthritis if he/she has satisfied at least four of these seven criteria. Criteria 1 through 4 must have been present for at least six weeks.

Patients with two clinical diagnoses are not excluded. Designation as classic, definite, or probable rheumatoid arthritis is not to be made.
BAKER’S CYST AND BURSITIS

There are numerous bursae around the knee, any of which can get inflamed or infected. The two main bursae usually affected are the pre-patellar bursa, overlying the patella, and the infra-patellar bursa, located over the tibial tuberosity. The pre-patellar bursa tends to become inflamed after prolonged “forward” kneeling. Pre-patellar bursitis, also known as ‘housemaid’s knee’ is common in trades such as roofers and carpet fitters. Infra-patellar bursitis, also known as ‘clergyman’s knee’ is common with more erect kneeling.

The pre-patellar bursa may also become infected, which can lead to its being confused with septic arthritis. An infected pre-patellar bursa tends to have a more localised swelling and erythema, and the knee joint movements tend to be well preserved. Should there be any diagnostic concerns, advice from a musculoskeletal specialist should be sought at the earliest opportunity, as previously stated.

Conclusion

The acutely swollen knee is a common presentation in primary and secondary care in both the civilian and military environment. However, with a generally younger and more active military population the incidence is probably much higher. This article has endeavoured to review the differential diagnoses and initial management of patients presenting with acute or recent onset swelling of the knee in the absence of trauma. The overarching aim is to raise awareness of these conditions and ultimately lead to earlier evaluation and treatment. We hope it will act as a guide or aide memoire to the medical professionals of the Royal Naval Medical Service, wherever they may be providing care.

To be continued. Please see Part Two in the next issue of the JRNMS.

References


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