

Monarthritis: an unusual presentation of renal cell carcinoma

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Abstract

Two cases of acute monarthritis secondary to asymptomatic renal cell carcinoma are described. This association has not previously been reported. The patients were initially thought to have a septic arthritis, but hot spots were seen on isotope bone scans and biopsy samples showed secondary neoplasms, which were later confirmed to be a result of renal cell carcinomas. The value of cytological examination of synovial fluid when there is clinical doubt as to the cause of a joint effusion is shown.

Acute monarthritis is a clinical emergency and requires a prompt diagnosis. It is essential to exclude a septic arthritis, though crystal deposition disease, reactive arthritis, or an initial presentation of what later may be a more generalised inflammatory arthropathy are other common causes of such presentations. Rarer causes of acute monarthritis include haemarthrosis and diseases such as pigmented villonodular synovitis, a malignant disease of the synovium. A secondary malignant deposit in a joint has not yet been reported.

We describe here two patients who presented within a short time of each other. These patients initially appeared to have a septic arthritis but were subsequently found to have solitary secondaries from a renal carcinoma.

Case reports

PATIENT 1

A 57 year old previously fit man presented with a four month history of a painful left ankle and was unable to bear his weight fully. His past medical history included rheumatic fever as a child without any cardiac sequelae and a recent episode of diarrhoea without any blood or mucus. He had no other systemic symptoms.

He was pale and pyrexial but a general examination was otherwise normal. There was swelling around his left ankle with marked erythema, warmth, and tenderness. All movements of the left ankle were excruciatingly painful.

The results of investigations were as follows: haemoglobin 104 g/l; erythrocyte sedimentation rate 90 mm in the first hour; white cell and platelet counts normal; blood urea, electrolytes, and liver function tests normal; serum acid phosphatase, plasma protein electrophoresis, and urine for Bence Jones protein negative on several occasions; plasma glucose 5.6 mmol/l; autoantibody and rheumatoid screen negative;

C reactive protein 55 mg/l (normal range 5-10); urine microscopy showed no evidence of haematuria; bacteriological and acid fast bacilli cultures were negative; blood cultures were sterile on several occasions; chest radiograph normal; and electrocardiograph normal.

The left ankle was aspirated and the joint fluid showed a total cell count of $36.4 \times 10^9/l$ (0.64 polymorphonuclear leucocytes; 0.36 lymphocytes). Gram's stain was negative.

A few pus cells and large pleomorphic cells with abundant cytoplasm were also detected, though no specific comments about malignant cells were made. No crystals were detected in the synovial fluid. Ordinary bacteriological cultures and cultures for acid fast bacilli were negative.

A radiograph of his left ankle showed an erosive lesion in the left talocalcaneal joint. Radiographs of the lumbosacral and sacroiliac joints were normal. An isotope bone scan showed a solitary hot spot on the left talocalcaneal joint.

The biopsy sample from the left talocalcaneal joint showed a partially necrotic tissue composed of fibrous tissue, cellular neoplasm, and foci of inflammatory cells. The neoplastic cells were large with clear vacuolated cells and large eosinophilic cytoplasm suggesting that the primary site was either the bronchus or kidney.

Subsequent investigations included computed tomography scans of the chest and abdomen. The computed tomography scan of the chest was normal; the abdominal scan, however, showed a large retroperitoneal mass associated with the right kidney with widespread infiltration into the perinephritic tissue, suggesting renal cell carcinoma.

He was treated with local radiotherapy to the ankle joint and the right kidney. He was not considered for total nephrectomy as the tumour had spread extensively into the perinephric tissue. He subsequently underwent tumour embolisation as a palliative procedure. His joint pain improved after local radiotherapy, and he was mobile with the help of a stick.

PATIENT 2

A 58 year old woman was admitted with a one week history of a painful right hip. She had primary generalised osteoarthritis for which she received intermittent treatment with non-steroidal anti-inflammatory drugs. She had previously had multiple sclerosis and primary hypothyroidism, for which she had been treated with regular thyroxine replacement. She had no other systemic symptoms and was reasonably fit.

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On examination she was overweight and had a temperature of 38.6°C. General examination was otherwise normal. Examination of the right hip showed gross limitation of movement in all directions as a result of pain.

Investigations showed haemoglobin 126 g/l and an erythrocyte sedimentation rate of 78 mm in the first hour; the white cell count was $18.6 \times 10^9/l$ and platelets were within the normal range. Her liver function tests and serum biochemistry were normal on several occasions. The autoantibody screen, including rheumatoid factor, was negative. Urine microscopy, culture, and blood culture, repeated on several occasions, did not grow any organisms.

A chest radiograph showed unfolding of the aorta but no other abnormality was detected. An electrocardiograph showed slow atrial fibrillation with a rate of 98 per minute. A radiograph of her hip joint showed a moderate degree of osteoarthritic changes and her lumbrosacral spine showed mild degenerative bone disease.

In view of the increased white cell count, pyrexia, and painful right hip, a provisional diagnosis of septic arthritis was made and the right hip joint was aspirated. Only a few millilitres of fluid was obtained. The synovial fluid showed a total cell count of $37.74 \times 10^9/l$, 0.72 polymorphonuclear leucocytes, 0.28 lymphocytes, and no organisms were seen on Gram's stain. Ordinary bacteriological cultures and cultures for acid fast bacilli were negative. Crystals were not detected in the synovial fluid. An isotope bone scan showed a hot spot on the superior margin of the right acetabulum.

An open biopsy sample was taken from the hip joint. The culture of the histological tissue was negative but microscopic examination suggested the possibility of a secondary deposit from either the kidney or ovary. A computed tomographic scan of the abdomen was then performed and it showed the presence of a mass in the left kidney with significant local spread and involvement of the para-aortic lymph nodes. The mass was later confirmed to be a renal cell carcinoma.

Discussion

Acute monoarthritis is a common rheumatological emergency and requires rapid diagnosis and treatment. It is often the result of an acute septic arthritis or an acute crystal arthropathy, although the two can occur in combination. A comprehensive review of published work over the last two decades did not find any reports of acute large joint malignant synovitis due to secondary malignant deposits.

Most skeletal malignancies are metastatic in nature rather than primary tumours and in a small percentage of patients the site of the primary tumour is never discovered.¹⁻⁵ The usual skeletal sites of metastases are the axial or proximal appendicular skeleton in patients older than 40 years, whereas primary bone

tumours occur in the appendicular skeleton in younger patients.⁶ Approximately 10% of metastatic bone lesions are solitary;⁷ in one series 35% of the solitary lesions identified on bone scans were secondary to primary genitourinary malignancies other than prostate.⁸ In a review of patients with renal cell carcinoma only 5% presented with metastatic bone disease and none of these had secondary deposits in the joints.⁹

The diagnosis of metastatic bone disease is aided when there are multiple areas of increased isotope uptake on a bone scan. The finding of an isolated area of increased uptake, however, requires further investigation. Although plain radiographs may suggest a diagnosis, a bone biopsy specimen is essential to achieve a histological diagnosis.¹⁰ Where, however, joint fluid is present, cytological examination might be of some help. Although this was not so in our patients, it is interesting that pleomorphic cells were seen in patient 1 and were similar morphologically to those seen later in the biopsy sample.

Where there are clinical doubts as to the cause of a joint effusion it is constructive to carry out a cytological examination on the synovial fluid or to save a small amount for later analysis. Early examination of synovial fluid in this way might avoid a later open or closed bone biopsy and might lead to a faster diagnosis.

The presentation of these two patients with acute malignant synovitis due to solitary secondary deposits in the joint questions whether such associations are rare or whether they are under reported. We suggest that such cases are both rare and under reported.

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