

## CASE FOUR

**Short case number: 3\_19\_4**

**Category: Musculoskeletal System & Skin**

**Discipline: Orthopaedics**

**Setting: General Practice.**

**Topic: Joint pain\_Gout [SDL]**

Case
<p><b>Carmello Abruzzi</b> is a 55 year old man who presents with sudden onset of pain in his right big toe. He has had similar episodes of pain before, however this has been the worse thus far. He has not been able to walk without pain and his toe feels hot and is swollen. He has been unable to wear his usual work shoes and has had to wear slippers today. Paracetamol has not helped relieve the pain.</p>

Questions
<ol style="list-style-type: none"><li>1. What are the key features of the history in the assessment of Carmello's toe pain?</li><li>2. What are the key features of examination that assist in differentiating the possible causes of a painful swollen joint?</li><li>3. Your provisional diagnosis is acute gout, Carmello asks you what causes gout; detail your explanation outlining the underlying pathophysiology of gout.</li><li>4. How does the pathophysiology of gout differ from pseudogout?</li><li>5. Carmello mentions that his father had gout, but he had large lumps on his elbows, explain the nature of chronic gout and the underlying pathophysiology of gouty tophi.</li><li>6. In your investigation of Carmello you organise an x-ray, demonstrate the radiological features of acute and chronic gout.</li><li>7. What further investigations would you do and why?</li><li>8. Briefly summarise the medications that are used in the management of gout and their mechanisms of action and potential side effects.</li><li>9. Carmello often enjoys a couple of glasses of red wine at night, he asks if he needs to change his alcohol intake or diet in any way, what would you recommend and why?</li></ol>

### Suggested reading:

- Solomon L, Warwick DJ, Nayagam S. Apley's Concise System of Orthopaedics and Fractures. 3<sup>rd</sup> edition. Danvers: CRC Press; 2005.
- Kumar P, Clark ML, editors. Kumar & Clark's Clinical Medicine. 8<sup>th</sup> edition. Edinburgh: Saunders Elsevier; 2012.
- Australian Doctor, 21 Aug 2009 Gout an other crystal Arthropathies: (Perrera, Brook, Tymns) Part 1
- <http://search.ebscohost.com.ipacez.nd.edu.au/login.aspx?direct=true&db=anh&AN=108379842&site=ehost-live&scope=site>
- Australian Doctor, 21 Aug 2009 Gout an other crystal Arthropathies: (Perrera, Brook, Tymns) Part 2 <http://search.ebscohost.com.ipacez.nd.edu.au/login.aspx?direct=true&db=anh&AN=108379843&site=ehost-live&scope=site>

### Question 1

**What are the key features of the history in the assessment of Carmello's toe pain?**

Acute gout:

- Sudden onset of acute pain that lasts for 1 to 2 weeks
- Often spontaneous but may be triggered by minor trauma, operation, alcohol, or unaccustomed exercise
- Common sites:
  - metatarsophalangeal joint of the big toe (75%)
  - ankle joint
  - finger joints
  - Knee Joint
- During the attack fever may be present.

Chronic gout:

- Polyarticular gout
- Tophi may form around joints and often also in the pinna of the ear and with time may ulcerate and discharge
- Joint stiffness and deformity as a result of joint erosion
- Renal damage due to deposition of urate crystals in the renal parenchyma
- Urate urolithiasis occurs in 10%

### Question 2

**What are the key features of examination that assist in differentiating the possible causes of a painful swollen joint?**

The commonest sites for acute gout is the metatarsophalangeal joint of the big toe, the ankle, the finger joints and the olecranon bursa. The skin looks red and shiny and there is considerable swelling. The joint feels hot and extremely tender.

The differential diagnosis includes:

- Cellulitis (not localised to joint, spreading erythema over skin)
- Septic bursitis
- Polyarticular gout that affects the fingers may be mistaken for rheumatoid arthritis
- Bunion
- Chondrocalcinosis - pseudogout
- Seronegative arthritis
- Septic arthritis

### **Question 3**

**Your provisional diagnosis is acute gout, Carmello asks you what causes gout; detail your explanation outlining the underlying pathophysiology of gout.**

There is an association between serum uric acid levels and risk of gout. The use of hypouricaemia medication results in a greater than 75% risk reduction of gout, reaffirming a causal relationship. Gout is caused by the deposition of monosodium urate crystals in tissues as a consequence of prolonged hyperuricaemia in both men and women, on average two decades after initial increases in serum urate concentration.

Hyperuricaemia may be due to overproduction or under excretion of uric acid. Primary hyperuricaemia may be related to increased endogenous production or renal hypo excretion unrelated to therapeutic or disease processes.

Secondary hyperuricaemia is excessive urate production or diminished renal excretion as a result of renal disease, toxins or dietary components.

Uric acid is weakly acidic and at physiological PH exists predominantly in ionized urate form. Increased concentration of urate in physiological fluids leads to super saturation setting the stage for crystal formation and deposition. A gout flare is caused by monosodium urate crystals triggering a leukocyte inflammatory response.

### **Question 4**

**How does the pathophysiology of gout differ from pseudogout?**

Pseudogout, (calcium pyrophosphate dehydrate deposition disease) is thought to be due to the generation of pyrophosphate in abnormal cartilage by enzyme activity at chondrocyte surfaces. It combines with calcium ions in the matrix, where crystal nucleation occurs on collagen fibres. The crystals grow into microscopic 'tophi' which appear in the cartilage matrix and in fibrocartilaginous structures such as the menisci of the knee and intervertebral discs. From time to time CPPD crystals are extruded into the joint, where they excite an inflammatory reaction similar to gout.

### **Question 5**

**Carmello mentions that his father had gout, but he had large lumps on his elbows, explain the nature of chronic gout and the underlying pathophysiology of gouty tophi.**

Patients who develop chronic gout usually are those whose hyperuricemia is not controlled. Tophi appearing within the first two years of gout are extremely rare and patients have usually suffered from gout for at least 10 years before tophi develop. The tophi are collections of crystals that form deposits in soft tissue, joint, bone and tendons. They cause erosion and destruction of the bone, and cause damage that may lead to crippling.

## **Question 6**

**In your investigation of Carmello you organise an x-ray, demonstrate the radiological features of acute and chronic gout.**

*Acute gout:* x- ray shows only soft tissue swelling

*Chronic gout:* asymmetrical, punched out ‘cysts’ in the juxtaarticular bone, joint space narrowing and secondary osteoarthritis.

## **Question 7**

**What further investigations would you do and why?**

Investigations

- Serum urate - may be raised. (in gout serum urate levels may be normal during an acute attack and thus the optimal time for measurement is about two weeks after a flare resolves )
- Aspiration and polarized light examination of synovial fluid - shows negatively birefringent crystals
- Leucocytosis and raised ESR and CRP during the acute attack
- other tests to consider
  - assessment of other CV risk factors - fasting lipid profile and glucose
  - depending on the clinical scenario - FBC, blood film, B12 and TFT's as on occasion elevated serum urate levels may be seen in conditions such as lymphoproliferative and myeloproliferative disorders, B12 deficiency, lead exposure and hypothyroidism

## **Question 8**

**Briefly summarise the medications that are used in the management of gout and their mechanisms of action and potential side effects.**

Non Pharmacological Treatment

- consider stopping or reducing diuretic treatment
- reconsider aspirin
- reduce alcohol intake
- reduce intake of purine-rich foods

Pharmacotherapy in acute gout:

- NSAIDS/COX 2 inhibitors +/- PPI
  - If no contraindications, NSAIDS at optimal dosing are the drug of choice and will result in rapid relief.

- Cobicine
  - Also effective but a slower onset of action
- Corticosteroids
  - intraarticular, IV or oral. Systemic therapy is effective treatment for acute flares only

Prophylactic allopurinol and uricosurics:

- are used for chronic gout only
- are not effective in acute attack
- are not used in an acute attack because they may prolong it indefinitely

**Question 9**

**Carmello often enjoys a couple of glasses of red wine at night, he asks if he needs to change his alcohol intake or diet in any way, what would you recommend and why.**

If you suffer from gout you will benefit in the long term from healthy changes to your lifestyle.  
Suggestions include:

- Maintain a healthy weight
- Limit wine, beer and other alcohol
- Drink plenty of non-alcoholic fluids, especially water
- Exercise regularly
- Treat high blood pressure
- Cut down or eliminate certain foods from the diet, such as liver, anchovies, shellfish and meat extracts
- Protect painful joints in bed. During a painful episode a 'cage' or bed cradle to lift the weight of bedclothes off the feet may be helpful.