Gout

May 25, 2015 | 0 | onthepods,rheumatology

James talks to Associate Professor Neil McGill about the management of gout on the wards. Gout is a crystal-induced disease driven by urate crystals forming in the joints.

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About Dr Neil McGill

Dr McGill is a Clinical Associate Professor in the Department of Medicine at the University of Sydney and a Consultant Rheumatologist at Royal Prince Alfred Hospital. He runs a busy general rheumatology practice and has a keen interest in teaching. He has had a long term interest in crystal-induced arthritis, stimulated initially by working with Professor Paul Dieppe in Bristol. Neil was the inaugural Chair of the Synovial Fluid Quality Assurance Program (within the RCPA Quality Assurance Program) in 1997 and he has continued in that role subsequently. He has contributed to many original publications and has written reviews in peer-reviewed journals in the field of crystal-induced arthritis.

Gout

With Associate Professor Neil McGill, Consultant Rheumatologist at Royal Prince Alfred Hospital, New South Wales, Australia

Case

A 60-year-old male with a history of excessive alcohol use and hypertension treated with diuretics presents with acutely swollen and painful ankle. He denies a history of trauma but does have a history of gout.

– 1. Can you give us a quick overview of gout? Pathogenesis of Gout

- Gout is a crystal induced disease – urate crystals drive the disease
- In order for urate crystals to form, hyper-uricaemia is usually required to be present
    - Saturation level of urate tends to occur at plasma urate concentrations of 0.41 mmol or higher
- Crystal formation is a slow process – occurring over months to years and usually doesn't cause pain
- Only a minority of people with hyperuricaemia (about 20%) will develop urate (monosodium urate monohydrate) crystals

- Favourable sites for precipitation of crystals:
    - Joints provide a favourable template for crystal formation
    - Crystals preferentially form in cooler sites hence more commonly in peripheral joints
- Painful and swollen joints are usually due to interaction between crystals and the immune system
- The inflammatory process often settles down on its own – takes a couple of weeks, but if treated will resolve faster
- The treatment does not clear the crystals, it only stops the inflammatory process

2. Outline your assessment approach by the bedside

- **History**
    - Risk factors in this scenario
        - Male
        - Alcohol use
        - Hypertension
        - Diuretics – thiazide especially (impair renal clearance of urate)
    - Other risk factors
        - Maori/Polynesian – reduced urate clearance
        - Renal impairment – reduced urate clearance
        - Alcohol – impairs renal clearance of urate
        - Other drugs – thiazide, anti-TB drugs, cyclosporine
        - Obesity
- **History findings consistent with gout**
    - Rapidity of onset is a useful point on history – often happens faster than septic arthritis
    - Previous history of gout – need to be careful to elicit how previous history of gout was diagnosed – whether there was joint aspiration etc.

History of acutely swollen joint in classical joints (e.g. 1st MTPJ i.e. 'podagra') that cleared with anti-inflammatory medications is strongly suggestive

- **Examination**
    - Careful search for tophi – often small tophi can be found on the ear, toes, fingers
    - Can put needle into a tophus and take to lab – need only tiny quantity such as within the bevel of needle

3. What is the difference between gout and pseudo-gout?

- Crystal involved is the main difference, however the basic principle of immune activation to crystals within the joint is the same
- Pseudo-gout – calcium pyrophosphate dihydrate crystal deposition disease
- Unlike gout which occurs in the context of hyperuricaemia, pseudogout occurs in the setting of normal calcium and phosphate levels systemically
- Increasing age is associated with increasing likelihood of forming CPPD crystals
- Can see calcium pyrophosphate on X-ray of joint – chondrocalcinosis = calcification of hyaline or fibrocartilage visible on xray

4. Management of gout?

- Depends largely on the co-morbidities of the patient
- If healthy otherwise, no significant co-morbidities
    - Full dose NSAID is sufficient such as meloxicam, indomethacin, etc.
- If contraindications to NSAIDS due to peptic ulceration, renovascular disease, etc. then need to consider alternative regimens
- Corticosteroids
    - Can be given as an intra-articular injection
    - Can prescribe oral prednisone if patient is reliable with good follow up
    - If patient not reliable – can give tetracosactrin depot (Synacthen depot) – to give patient endogenous boost of corticosteroids for next 7-10 days
    - Need to be careful if patient has diabetes – monitor BSLs
- Colchicine
    - Tricky drug to titrate
    - Not often the most effective drug
    - If used – use low dose regime – 1mg initially, followed by 500mcg one hour later and then no more for 24 hours
    - Just as effective as previously used high dose regime
    - Often colchicine is added as adjunct to acute therapy (NSAIDs, corticosteroids) as bridging to long term urate lowering therapy such as allopurinol

5. What about long term therapy of gout? When should we be starting allopurinol or urate lowering therapy drugs?

- This applies to all urate lowering therapies such as allopurinol, probenicid or febuxostat
- If patient presents with acute attack and is already on urate lowering therapy, these should NOT be stopped and should be continued at the same dose
- Urate lowering therapies should NOT be started during acute attacks – may make acute attack worse
- Urate lowering therapy is a life-long decision and should not be made during the acute attack and therefore should be discussed at follow-up
- Who should use urate lowering therapy
    - Ensure diagnosis is secure, preferably crystal proven
    - Presence of tophi
    - Chronic symptoms between attacks
    - Erosions on X-ray of joint
    - Multiple attacks
    - Proven gout and renal insufficiency
- Target levels of urate
    - No tophi < 0.36 mmol/L
    - Tophi < 0.30mmol/L

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