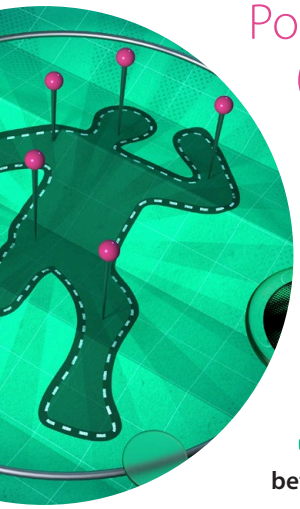

Polymyalgia rheumatica (PMR) – look before you leap

The following questions can be used as discussion points for peer groups or self-reflection of practice. It is strongly recommended that the following article is read before considering the questions.

- "**Polymyalgia rheumatica (PMR) – look before you leap**"

Polymyalgia rheumatica (PMR) is an inflammatory rheumatological condition that causes a specific pattern of joint pain and morning stiffness, and almost never occurs in people aged < 50 years. A diagnosis of PMR is made clinically based on recognition of a history of characteristic symptoms, with a raised CRP (or ESR if CRP is normal), exclusion of other conditions which may mimic the history (e.g. rheumatoid arthritis) and a rapid response to oral corticosteroid treatment.

Giant cell arteritis (GCA), a form of vasculitis, has a strong association with PMR and up to one-fifth of people with PMR will develop GCA at some point. Therefore, a high suspicion for GCA is needed before and during treatment for PMR to prevent ischaemic complications, e.g. permanent blindness. If GCA is suspected (e.g. a patient with new onset or type of headache, temporal artery or scalp tenderness, jaw claudication and visual symptoms) refer the patient for an urgent temporal artery biopsy (or discuss with the rheumatology service according to local protocols) and initiate high dose oral corticosteroids as soon as possible.

Assessment of the patient's response to treatment for PMR (usually prednisone) should primarily be based on their clinical response, e.g. ability to perform movements and tasks that were previously impaired (such as getting out of a chair). Symptoms generally improve rapidly after treatment initiation, i.e. within a few days to one week. An inadequate response to prednisone within seven to ten days is uncommon and if this occurs, the diagnosis should be reconsidered. If clinical suspicion for PMR remains, consider trialling a higher prednisone dose after discussing with the local rheumatology service.

Monitoring CRP (or ESR) levels is often helpful when assessing a patient's response to prednisone; these should normalise within two to four weeks, however, inflammatory marker levels may not always correlate with the patient's symptomatic response. Once there is adequate improvement in the patient's symptoms, slowly and progressively taper the corticosteroid dose. The rate of taper should be done primarily in response to the patient's symptoms, not CRP (or ESR) results alone. Relapses are not uncommon during this time and if they occur, increase the dose back to pre-relapse levels.

PMR is usually a self-limiting condition, and while the aim is to stop corticosteroid use as soon as possible, the average duration of treatment is one to two years. Long-term treatment decisions are individualised, taking into consideration the adverse effects associated with prolonged corticosteroid use, e.g. osteoporosis, diabetes, gastritis, adrenal insufficiency. For patients who require an alternative to corticosteroids, treatment decisions should be made following consultation with a rheumatologist; methotrexate is an option.

Questions for discussion:

1. Symptoms and signs of PMR often overlap with other similar conditions. How do you typically differentiate the cause of these symptoms and what is your threshold for suspecting PMR, e.g. presence of risk factors such as age, laboratory results? Prior to reading this article, were you aware that almost all people affected are age > 50 years and of European ethnicity?

2. In your experience, how often do you find that patients with PMR do not have elevated CRP or ESR levels during the diagnostic work-up? If acute phase response markers are not elevated, but you still suspect PMR based on the patient's symptoms and signs, what are your next steps?

3. How confident do you feel in being able to identify when a patient is presenting with GCA? What factors do you consider when making the decision to refer for an urgent temporal artery biopsy and initiate high dose oral corticosteroids? Have you ever had a patient develop GCA during treatment for PMR? If so, what were their symptoms and signs?

4. When treating a patient for PMR, what dose of prednisone would you generally initiate them on? Would this change based on co-morbidities, e.g. BMI, frailty, or do you stick to a standard dose? Most patients respond rapidly within days to corticosteroid treatment, would you agree with this based on your experience?
5. What is your strategy on determining an appropriate dose tapering regimen? How long do you usually keep patients on corticosteroids for? How do you approach patients who struggle to make the final corticosteroid dose reduction and require treatment for years, e.g. do you add weekly methotrexate, reconsider the diagnosis, refer?
6. Ideally, all patients taking long-term corticosteroids for PMR should be reviewed periodically to assess response to treatment, adjust dose of corticosteroid as required, and monitor for any symptoms or signs of relapse, corticosteroid-related adverse effects or the development of GCA. Do you actively recall patients or review at the next available opportunity? How do you find that patients usually tolerate treatment and what adverse effects from corticosteroids would you say are most commonly experienced?