

GP & AHP Newsletter



UPDATE FROM THE LONDON CLINIC

FEBRUARY 2021

Dear Colleague

Welcome to our first newsletter of 2021, a fresh start! We are excited to work with you this year to provide a renewed and united effort to move forward and reinvigorate healthcare, in spite of the ongoing challenges.

The good news is that administration of the vaccine is well underway, including here at The London Clinic, with over 76% of staff now vaccinated.

In this newsletter we are pleased to share a news feature from Dr Stephanie Kaye-Barrett, Consultant Rheumatologist and General Physician. Read her article on The Immune System and Organ Damage on page two.

- We are collaborating with and supporting the NHS during this time, whilst continuing to be open to all private practice across all priorities
- A number of new services are launching soon, including:
 - MSK trauma and rehabilitation
 - A fracture clinic
 - An allergy clinic
- We illuminated our main hospital in orange and blue in support of [#WorldCancerDay](#)
- The next edition of The London Clinician focuses on MSK and will be available next month. The previous edition focused on oncology and can be read [here](#).

We hope you enjoy this newsletter and as always we welcome your feedback.

Akta Chauhan



NEW Consultants to join the The London Clinic this month

- » **Mr Jahangir Ahmed** Consultant in Head & Neck, Thyroid, Airway and ENT Surgery
- » **Mr Jagwant Singh** Consultant Orthopaedic, Trauma & Specialist Upper limb Surgeon
- » **Mr Gerassimos Lascaratos** Consultant Ophthalmic Surgeon

- » **Mr Chadwan Al Yaghchi** Consultant Laryngologist and Ear, Nose and Throat Surgeon
- » **Dr Shahid Jawed** Rheumatologist
- » **Dr Timothy Watts** Allergist



Register for webinar events by contacting
events@thelondonclinic.co.uk

Upcoming Events

Thursday 18 February
GP Webinar

12:30pm – Dr Arshad Rather,
Care of the elderly

Tuesday 23 February
GP Webinar

12:30pm – Professor Francis Vaz
Neck lumps

Wednesday 24 February
GP Webinar

12:30pm – Mr Gordan Grahovac
Disc prolapse & tips for virtual examinations

Wednesday 24 February
GP Webinar

12:30pm – Davide Lanfranco
Lower back pain: Separating facts from fiction



INFLAMMATION

The Immune System and Organ Damage

Dr Stephanie Kaye-Barrett, MBChB, MD, FRCP.
Consultant Rheumatologist and General Physician.
Past President of the Royal Society of Medicine.
www.drstephaniebarrett.com

My aim is to adopt a ‘multi stranded’ approach to the topic of inflammation, which has been shown to increase mortality due to cardiovascular complications.

Essentially, **rheumatoid arthritis, psoriatic arthritis and gout** are ‘smoking guns’ and potential killers, unless we, as clinicians, diagnose them early and treat the underlying inflammation, as seriously and rigorously as we do angina in cardiac patients.

Rheumatoid arthritis (RA) and psoriatic arthritis (PsA) both cause structural joint damage unless treated, with disease modifying agents (DMARDs), within three months of detection of joint inflammation. Besides this, patients with untreated RA have twice the risk of myocardial infarction and angina. The statistics are similar for PsA. Women with early menopause are at even greater risk.

Previously, **gout** was thought to be an excruciating and acutely debilitating condition, but not potentially life threatening. However, it has been demonstrated that gout shares the same **increased cardiovascular risk** as the other inflammatory joint diseases. In addition, asymptomatic **hyperuricaemia** has been shown to be an **independent risk factor for cardiovascular disease**. Possibly the presence of undetected gout crystals, stored within the soft tissues, creates an environment of constant low-grade, systemic inflammation.

These three diseases share the common denominator of raised levels of C-reactive protein (CRP), a marker of inflammation also found in smokers and hypertensive patients. There are direct links between high Hs CRP levels and heart disease, stroke and sudden cardiac death (2-3 times greater than in the normal population).

Thus, we must strive to detect and treat gout and RA/PsA early and rigorously, and liaise with our cardiology colleagues and rheumatologists on the best management of the inflammatory process. Treatment and modification of all additional cardiac risk factors such as hypertension, hyperlipidaemia, obesity and cessation of smoking should occur concurrently.

Non-steroidal anti-inflammatory drugs should be used sparingly and for short periods, as they also carry increased cardiovascular risk. **Gout** should be treated early with **allopurinol** (after second episode), striving to achieve uric acid levels, at or below 300mmol/l, as far as possible and **using colchicine 500mcg bd-tds for six weeks whilst introducing the allopurinol incrementally and slowly over three weeks to reach 300mg/day in the third week**. In RA/PSA urgent referral to a rheumatologist and commencement of **DMARDs** and **anti TNF drugs/biologics**, both prevent joint damage and are **cardioprotective**, as long as the inflammation (judged on patient history, examination, CRP and peripheral joint ultrasound), is under control.

Problems with early **detection of gout** lie in the **normal or low uric acid levels**, which are frequently found in acute gout. The presentation is key, with acute hot red and tender joints where there is no evidence of infection (normal WCC and lack of fever). Patients often respond very well to **intramuscular depomedrone 120mg and this can be followed by colchicine 500mcg tds with the introduction of allopurinol 100mg/day in week one, 200mg/day in week two and 300mg per day, thereafter**.

Where patients are intolerant of allopurinol, or where febuxostat is unsuitable, **fenofibrate 200-267mg may be used**. This is a uricosuric drug, which is well tolerated and used frequently by rheumatologists as a further alternative to allopurinol.

COVID-19, studies have revealed that **dexamethazone** is an effective drug, used to control the ‘**COVID-19 cytokine storm**’, the over exuberant inflammatory response produced by the immune system, in response to **Sars-CoV-2 infection**, which is the greatest cause of mortality.

Corticosteroids are currently the first line in treatment of the ‘**COVID-19 cytokine storm**’, or **hyperinflammation**, which causes severe derangement of clotting systems and multiple organ failure. It is characterised by extremely high levels of CRP, LDH and ferritin. Lymphopenia is also a common finding. In a recent study from the University of Southampton, high levels of five cytokines have been found, **IL6, IL8, IL1beta and IL-33, which were associated with a greater chance of needing ICU, artificial ventilation and dying**. Tocilizumab (usually used for RA) has just been shown to reduce mortality of COVID-19 ICU patients by 24%. A combination of an IL6 inhibitor, tocilizumab and steroids produced even greater reductions of mortality, compared to steroids alone. Colchicine is now being investigated as a therapeutic option in Covid hyperinflammation.

Some ‘long COVID’ patients continue to show a modestly raised CRP, together with muscle pain and fatigue (polymyalgia symptoms), long after the viral infection has left the host. If accompanied by a raised CRP and after screening for infection, diabetes etc., a trial of low dose steroids 15mg/day and tapering to 7.5mg/day may be used as a therapeutic trial and as treatment in the short term. It has been found to be of anecdotal benefit in a small number of patients. We will learn more as the ‘long COVID’ clinics are set up.

LEARNING POINTS:

- 1) Identify and treat inflammatory diseases as soon as possible and bring them under control
- 2) Monitor the CRP closely together with other important signs of inflammation
- 3) Corticosteroids are of crucial importance in the treatment of the ‘cytokine storm’
- 4) Immune modulators/biological agents such as tocilizumab, and anti TNF drugs are being used in RA, PsA and now the COVID-19 cytokine storm to control inflammation and reduce mortality
- 5) Remember to treat gout in the long term to reduce hyperuricaemia, reduce crystal load and prevent excess cardiovascular deaths
- 6) Fenofibrate may be used as an alternative uricosuric agent in gout, where allopurinol or febuxostat are unsuitable, for long term prevention and control of the uric acid levels to 300mmol/l or below.