Five Things Every Physician should know about the Pharmacologic Management of Gout

Marc-Etienne Parent* and Patrick Liang

Department of Rheumatology, University Centre Intégré de Santé et de Services Sociaux de l’Estrie, Centre Hospitalier Universitaire de Sherbrooke, Canada

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*Corresponding author: Marc-Etienne Parent, Department of Rheumatology, Centre Intégré de Santé et de Services Sociaux de l’Estrie, Centre Hospitalier Universitaire de Sherbrooke, Canada, Tel: 1-819-346-1110; Email: Marc-Etienne.Parent@USHerbroke.ca

Abstract

1. Urate Lowering Therapy (ULT) is initiated alongside acute attack treatment.
2. ULT follows a treat-to-target approach.
3. Start low, go slow, but get somewhere.
4. ULT should not be interrupted.
5. Many drugs influence uricemia.

Keywords: Hyperuricemia; Gout; Urate Lowering Therapy; Pharmacology; Best Practice

Abbreviations: UTl: Urate Lowering Therapy; NSAIDs: Nonsteroidal Anti-Inflammatory Drugs; EULAR: European League Against Rheumatism

Short Communication

1. Urate Lowering Therapy (ULT) is initiated alongside acute attack treatment

Contrary to popular belief, the initiation of ULT during a gout attack does not prolong the attack [1]. However, the initiation/titration of ULT causes uricemia fluctuations, which can precipitate attacks. Thus, prophylaxis with low dose colchicine or non steroidal anti-inflammatory drugs (NSAIDs) should be continued until targeted uricemia is reached and ULT dosage is stable [2].

2. ULT follows a treat-to-target approach

ULT should be progressively increased to reach a specific target. The target, usually 360 or 300μmol/L, is determined by the severity of the disease (see Box 1). Maintaining excessively low uric acid levels (<180μmol/L) is not recommended in the long term [2].

3. Start low, go slow, but get somewhere

Only 5-34% of patients are using >300mg daily of allopurinol with as much as 72% not achieving their targets [3]. The recommended maximum allopurinol dosage, in the absence of severe renal disease [4], is up to 800mg daily. Despite this, if the target is not achieved alternative agents should be sought out, such as febuxostat [2]. Additionally, febuxostat use was recently shown to be secure even in patients with severe chronic kidney disease [5].

4. ULT should not be interrupted

Once the targeted uricemia is reached, a new steady state is created. If ULT is stopped during an attack, the additional uricemia fluctuation will further disturb this steady state, as well as create the need to re-introduce the ULT in the future. It is necessary, however, to re-evaluate the adequacy of ULT and to optimize it as necessary [2].

5. Many drugs influence uricemia

Reviewing the patient’s medication can prove useful. Aspirin, diuretics, and sildenafil can increase uricemia [6], while vitamin C, folic acid, and caffeine can decrease uricemia and the risk of recurrent gout [7]. Certain antihypertensive agents, such as calcium channel blockers and losartan, can aid in further reducing uricemia [2].

Table 1: 2016 European League against Rheumatism (EULAR) Recommendations for the Initiation of ULT and Treatment Targets (Category of evidence, Grade of recommendation) [2] (Table 1).
of urate-lowering treatment during an acute gout attack prolong the current episode and precipitate recurrent attacks: a systematic literature review. Rheumatol Int 36(12): 1747-1752.

<table>
<thead>
<tr>
<th>ULT indications [1b,A]</th>
<th>Serum uric acid target of under 360μmol/L [3,C]</th>
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<tr>
<td>Recurrent flares (&gt;1/year)</td>
<td>All patients</td>
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<td>Presence of tophi</td>
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<td>Young age (&lt;40 years)</td>
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<td>Cardiovascular comorbidities (hypertension, renal impairment, ischemic heart disease, heart failure)</td>
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<td>Serum uric acid target of under 300μmol/L [3,C]</td>
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<td>Tophi</td>
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<td>Chronic uratearthopathy</td>
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<td>Frequent attacks</td>
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Table 1: ULT: Urate Lowering Therapy.

References

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