Antiuratics

Antiurarics are substances that **reduce uricemia** and are used as a treatment of gout. Together with medically lowering the levels of uric acid in the blood, there are key measures in the treatment of gout. We distinguish between the therapy of **permanent hyperuricemia** (chronic gout) and **acute gout attacks**.

Therapy of chronic hyperuricemia

Based on the mechanism, we divide medications into **uricostatics** that block the synthesis of uric acid and **uricosurics**, which increase the excretion of uric acid.

Uricostatics

The most important representative of uric acid formation blockers is **allopurinol**. A new alternative, in case of allopurinol intolerance, is the oral non-purine selective inhibitor of xanthine oxidase, **febuxostat**[1].

Allopurinol

|thumb|right|300px|Synthesis of uric acid It is an isomer of hypoxanthine, which inhibits xanthine oxidase by competing with its substrate. Accumulating metabolites hypoxanthine and xanthine are simply excreted by the kidneys. It is well tolerated, but some allergic reactions or digestive problems might occur.

- **Indication**: primary and secondary gout; urate nephrolithiasis
- **Contraindication**: allergies; acute gout attacks; use of purine analogues (e.g. azathioprine), because of an increased risk of bone marrow depression

Uricostatics

They are secreted by the tubules and increase uric acid excretion by inhibiting tubular reabsorption. Their effectiveness decreases with renal insufficiency.

Main used substances

- ***Benzbromarone***: it is used in combination with azatioprine and its side effects include gastrointestinal problems, hepatotoxicity, and an increase in the effect of Vit-K dependent anticoagulants.
- ***Probenecid***: it is used more often in English speaking countries in combination with allopurinol (e.g. **allomaron**) and when used, it inhibits the secretion of penicillin or indomethacin.

Acute gout attacks

In acute manifestations of gout, therapy against **pain** and **inflammation** is essential. The release of lysosomal enzymes leukocyte phagosomes is largely responsible for the symptoms. That is why **colchicine** and non-steroidal anti-inflammatory drugs (the most effective for gout arthritis is indomethacin) are used as therapy. When NSAIDs and colchicine are contraindicated, systemic glucocorticoids are administered for 7-9 days.[1] For non-pharmacological therapy, local cooling and rest is recommended.

⚠ In the case the first detection of an acute attack, drugs that reduce the levels of uric acid are not used. They might trigger another attack or prolong an existing one. In case of an ongoing treatment, they are **not withdrawn.**[1]

Colchicin

It is an alkaloid working as a mitotic poison. It binds to tubulin, which depolymerizes and thus blocks the migration and mobility of neutrophil granulocytes. It relieves pain within 12 hours and is used p.o. for a maximum of 48 hours. In case of overdose, its toxic effects may even lead to death (from 15mg).

- **Side effects**: diarrhea and dehydration as signs of gastroenteritis; neuropathy; myopathy; an effect on hematopoiesis
- **Contraindication**: hepatic and renal insufficiency

Links

Related articles

- Arthritis uratica

References

1.

Used literature

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- HERDEGEN, Thomas. *Kurzlehrbuch Pharmakologie und Toxikologie : 328 Tabellen.* - edition. Thieme, 2010. pp. 218. ISBN 9783131422927.