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Current Treatment for Gout: A Review

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Abstract. Gout is a prevalent inflammatory arthritis resulting from the deposition of monosodium urate (MSU) crystals in tissues, typically characterized by severe pain and swelling at the affected joints. This review article aims to understand the reasons for the global rise in gout cases through examining the underlying mechanism and evaluating available treatments. Four treatments—NSAIDs, colchicine, glucocorticoids, and xanthine oxidase inhibitors—are evaluated based on mechanisms of action, commonly used drugs, and associated safety issues. NSAIDs, with ibuprofen, aspirin, and naproxen sodium as the most used drugs, exert their effect by inhibiting prostaglandin production from the COX enzyme, although their adverse effects of gastrointestinal disorders warrant attention. Colchicine has long been used to relieve joint pain and swelling by inhibiting microtubules, blocking NLRP3 activation, and reducing neutrophil recruitment. However, it has comparatively narrow therapeutic uses as high doses may lead to severe toxicity. Glucocorticoids relieve acute gout by inhibiting leukocyte recruitment and by suppressing inflammation and vascular permeability. Prednisone, methylprednisolone, and triamcinolone are used depending on disease severity and sites of onset. They are recommended only for short-term treatment due to the risks of ulcers. Lastly, xanthine oxidase inhibitors, allopurinol and febuxostat, focus on maintaining serum urate levels to prevent crystal formation. However, side effects include allopurinol leading to allopurinol hypersensitivity syndrome and febuxostat causing cardiovascular events. The major issues lie in drug safety and knowledge gaps. Future research should focus on eliminating side effects; professionals and patients should be educated on gout treatment and prevention.

Keywords: gout, treatment, drug analysis, hyperuricemia

1. Introduction

Gout, a common inflammatory arthritis disease that may occur sporadically in individuals, is characterized by sudden attacks of pain and swelling in joints. Reports from 2023 show gout prevalence to have increased by 63.44% over the past 2 decades, highlighting the need for awareness in gout treatment [1]. The reasons for the distress are hyperuricemia and monosodium urate (MSU). As byproducts of purine metabolism, uric acids are normally dissolved in blood and

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excreted by the kidneys. High levels of uric acid will then result in hyperuricemia, where they will form MSU crystals and deposit in joints, leading to inflammation. Although the swelling may subside, crystals can persist in the joint. Known risk factors for gout are primarily associated with uric acid, meaning diet, medication, and heredity are major contributors [2]. Furthermore, kidney diseases and high blood pressure can all lead to gout attacks, both of which would impede uric acid excretion [3]. Thus, treatment methods should aim to lower the serum urate level and dissolve crystal deposits. This review aims to analyze the conventional therapeutic strategies for different stages of gout, thus exploring the underlying reasons for the global increase in cases of gout.

There are two stages of gout: acute flares and established gout. Acute flares refer to sudden onsets of painful inflammation, usually happening on the foot and great toe. Since it is short-term, treatment methods aim to relieve immediate pain. Amongst available pharmacological options, NSAIDs, colchicine, and glucocorticoids are the most preferred due to their well-established efficacy.

Established gout treatment aims to resolve the underlying metabolic cause of gout-hyperuricemia-through maintaining serum urate levels and thereby preventing formation and promoting crystal dissolution.

2. Current treatment 1: Nsalds

2.1. Mechanism

Nonsteroidal anti-inflammatory drugs (NSAIDs) are a group of medications that are widely used for pain relief, fever mitigation, and managing various inflammatory conditions. NSAIDs carry out their function largely by inhibiting cyclooxygenase (COX) enzymes, which are crucial for prostaglandin synthesis. They can affect two isoforms of COX enzymes: COX-1 and COX-2. COX-1 is a constitutive enzyme that is present in most tissues and organs. It serves multiple functions, such as maintaining platelet activity and regulating renal blood flow. COX-2 is an enzyme that produces prostaglandins (especially PGE_2) and is primarily induced at the sites of inflammation [4]. Prostaglandins are lipid-derived signaling molecules that bind to receptors on cells and amplify pain signals and inflammation at the affected joint. This action is facilitated by a special prostaglandin transporter called PGT [5]. With the crystalisation of monosodium urate around the joint, prostaglandins are continuously produced at low levels by COX-1, while COX-2 expression is triggered by chemical signals, resulting in a surge of prostaglandin synthesis [5]. Therefore, by inhibiting COX enzymes, NSAIDs alleviate the inflammation and pain commonly within 3-5 days, along with swelling and redness around the problematic joint [6].

2.2. Treatment

Non-selective and COX-2 selective agents are the two major categories of FDA-approved NSAIDs [4]. Non-selective NSAIDs inhibit both COX-1 and COX-2 enzymes, while COX-2 selective NSAIDs are specific and target COX-2 only. The choice of NSAIDs should depend on different situations, as no single drug is inherently superior. Regardless of the NSAID chosen, treatment of acute gout flares usually requires higher doses [6].

Some typical examples of NSAIDs include:

Ibuprofen: A non-selective inhibitor that remains to be the most widely used drug due to its outstanding analgesic ability, even though its anti-inflammatory effects are less effective than other

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NSAIDs in the class [7]. The dosage for ibuprofen tends to be 1 to 2 tablets every 4 to 6 hours if symptoms persist. Intake should be no more than 1200mg per day [4].

Aspirin: A non-selective NSAID with stronger nausea and gastric adverse effects than ibuprofen [7]. However, it is preferred due to its strong anti-inflammatory abilities and broad therapeutic uses, for instance, treating cardiovascular disease and fever. Common dosages are 1 to 2 tablets every 4 hours, or 3 tablets every 6 hours, depending on specific symptoms; maximum intake of aspirin is 4000 mg per day.

Naproxen sodium: A non-selective inhibitor in NSAIDs. Although it does not affect alleviating disease progression, its duration of pain management is remarkable, lasting 8 to 12 hours. Daily maximum intake is 660mg [4].

In general, NSAIDs have several merits in clinical use, such as short onset time and relatively low cost [8]. Additionally, NSAIDs not only reduce pain by mitigating inflammation, but also exert effects through their intrinsic analgesic activity, which enables pain relief without waiting for inflammation to be fully controlled [6].

2.3. Safety

However, there are a few risks of NSAID usage that need to be carefully considered. First is a gastrointestinal issue. Prostaglandins protect the gastric mucosa in various ways. Thus, the inhibition of prostaglandin production in COX-1 might lead to gastric ulcers and gastritis, particularly in those with a history of gastric issues. Second is cardiovascular risk. The incidence of cardiovascular events would surge due to an increase in the usage of COX-2 inhibitors in NSAIDs. Some common adverse outcomes include embolic events, atrial fibrillation, and stroke [4,9]. In addition, NSAIDs can cause hemostatic disorders by disrupting the normal blood-clotting process, thereby elevating the risk of both external and internal bleeding [9]. This antiplatelet effect is specific to non-selective NSAIDs. While hemostatic disorders may pose a small effect on the general population, it is particularly dangerous for patients with a history of coagulation-related disorders such as hemophilia and thrombocytopenia [4].

Overall, non-selective NSAIDs inhibit a broader range of COX enzymes compared with COX-2 selective NSAIDs, which may result in a higher incidence of adverse effects [9].

3. Current treatment 2: Colchicine

3.1. Mechanism

Alkaloids are one type of naturally occurring compound that contains nitrogen. Made by various organisms such as fungi and plants, alkaloids are especially important because they include some of the most medically valuable components found in plants. An alkaloid, colchicine, is extracted from the plant Colchicum autumnale and is used to treat gout [10]. Colchicine is an anti-inflammatory therapy authorized by the FDA in 1961, used to treat acute gout. Firstly, colchicine would enter the cell and bind to tubulin dimers to prevent microtubule polymerization [11]. It suppresses monosodium urate (MSU) and calcium pyrophosphate (CPP) crystal-induced Nod-Like Receptor Protein 3 (NLRP3) inflammasome in macrophages, which inhibits innate immune responses. Therefore, it inhibits caspase-1 activation and subsequent IL-1b and IL-18 release. Thirdly, by altering adhesion molecule E-selectin distribution in endothelial cells and L-selectin expression in neutrophils, restraining the chemotactic agent leukotriene B4, and changing neutrophil deformability, colchicine decreases neutrophil adhesion and recruitment [12].

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3.2. Treatment

Colcrys is used to treat and prevent gout in adults, particularly suitable for those starting or planning to start urate-lowering therapy. Doses are 1.2mg taken once daily. It is contraindicated in patients who have taken NSAIDs within the past 30 days [13]. Compared with other medicines, Colcrys holds many benefits. In a study dedicated to studying Colcrys, data reported that in the first 48 hours of treatment, 73% patients who used it reported that pain in joints improved by 50% or more. A similar experiment conducted in 2017 showed a similar trend [12]. Moreover, it is also proven effective in preventing gout—especially in patients who started using urate-lowering therapy. In 2010, a clinical experiment found that the morbidity of gout is the lowest 6 months after using Colcrys for gout prevention [14,15]. However, the FDA recommends patients with liver dysfunction avoid concurrently taking it along with P-gp or strong CYP3A4 inhibitors. If patients are already taking these inhibitors, doses of Colcrys should be reduced or interrupted. Caution should also be taken when using Colcrys with weak CYP3A4 inhibitors, as this may cause risks of toxicity and an increase in the blood drug concentration of colchicine [14].

ColBenemid is a medicine that contains 0.5 mg colchicine and 500 mg probenecid. Approved by DESI, it is efficient to cure acute gout [16].

3.3. Safety

Lower doses are recommended for people who have renal injuries or who take other medicines that may cause drug interactions with colchicine [17]. People who have extremely serious kidney or liver dysfunction cannot use colchicine, because the kidney and the liver are involved in the metabolism of colchicine [14]. In these cases, the regular dose method is not recommended, since the incidence rate of gastrointestinal toxicity is high.

Colchicine has a narrow therapeutic index: excesses of 0.5mg/kg in dosing will cause death. Dosing of toxicity to as low as 3mg will also lead to death. Colchicine poisoning is characterized by cells stopping mitosis [17].

Patients with mild to moderate renal impairment do not require a dose adjustment. For patients having serious renal impairment, doses should be taken every once in two weeks. Patients receiving dialysis should regulate the dose to 0.6mg and treat it once every two weeks. Colchicine is not recommended for acute flare treatment in patients with kidney dysfunction [14].

4. Current treatment 3: Glucocorticoids

4.1. Mechanism

Glucocorticoids (GCs) have strong anti-inflammatory effects. They inhibit the recruitment and activation of critical blood cells, such as neutrophils and macrophages, which play an important role in increasing inflammation at the MSU crystal deposition site. Moreover, GCs reduce the production of some pro-inflammatory mediators and prevent the leakage of liquid and inflammatory cells into joint cavities, thereby relieving joint edema and the severe discomfort that prevents patients from bearing weight or moving the joint [18]. GCs are considered the first optimal therapy for gout [19].

4.2. Treatment

GCs are recognized as the first-line therapy [18]. Prednisone, a common oral administration medicine, is indicated for patients with many joints affected simultaneously or when patients have

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contraindications or intolerances to other medications(20-40mg/day) [20]. The standard dosage is 0.5mg/kg/day for 5-10days, followed by a gradual taper [18,21].

When combined with other medications, such as low-dose colchicine, the probability of gout decreases, plus renal function is not affected. Moreover, a study in 2004 showed that prednisone can be used in patients with systemic lupus erythematosus undergoing acute gout, with eight patients receiving different doses of prednisone [19].

Four emergency departments in Hong Kong did a study. They recruited 416 patients, and divided them into two groups, the first group used GCs(20-40mg/day), while another one used Indomethacin(50-100mg/day)In two hours, the resting pain of group of prednisone reduced by 10mm, they pain during movement is decreased by 45mm, while another group was the same. In 14 days, the resting pain of both groups reduced by 25mm, and pain during movement reduced by 45mm [22].

If other medications cannot control symptoms in 2-3days, or patients cannot accept oral drugs, methylprednisolone, a synthetic medium-acting glucocorticoid drug, can be used as a substitute for prednisone. It shows potent anti-inflammatory, anti-allergic, and immunosuppressive effects [23]. The recommended regimen is intravenous administration of 40-80mg/day for 1-3days, followed by oral therapy with gradual dose reduction. After intravenous administration, the swelling of the joint subsides within 12-24 hours, and the pain Visual Analogue Scale (VAS) score decreased from 7.5 ± 1.2 to 3.0 ± 0.8 ($p < 0.01$). Moreover, the total treatment duration in the intravenous group (7 ± 2 days) is shorter than that of the oral prednisone group (10 ± 3 days), with no difference in the 6-month recurrence rate (both 12%) [18,21].

4.3. Safety

High doses of prednisone (40mg/day) can lead to adverse reactions, such as renal function impairment and intestinal perforation. But some adverse reactions, such as common infections, have nothing to do with poor underlying kidney function [19]. GCs do far less harm to people with poor kidney function than NSAIDs. GCs do not affect glomerular filtration rate by inhibiting prostaglandin synthesis, thus avoiding the risk of kidney damage associated with NSAIDs [21]. For patients with acute gout, using GCs won't increase the risk of cardiovascular disease. Unlike NSAIDs, they don't have the risk of ulceration [18]. In conclusion, for patients with some special reasons, GCs are a safer option.

If a person uses GCs for a long period, he/she may develop osteoporosis, thus the risk of fractures will be significantly increased. If using this medication for over 3 months, bone density will decrease by 3-5% each year [18,21]. However, GCs cause far less harm to people with poor kidney function than NSAIDs and maintain the same cardiovascular and gastrointestinal safety advantages noted above [18,21]. In conclusion, for patients with some special reasons, GCs are a safer option.

5. Current treatment 4: xanthine oxidase inhibitors

5.1. Mechanism

As of current stages, the first-line long-term treatment is a strategy called urate-lowering therapy (ULT), which is a method that can effectively lower urate levels in the body, thus preventing gout attacks. As a commonly used method in urate-lowering therapy (ULT), xanthine oxidase inhibitors reduce hyperuricemia by blocking the production of urate. Inhibitors target the enzyme xanthine oxidase, which exacerbates gout conditions through facilitating purine breakdown—causing

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hypoxanthine to break into xanthine, then into uric acids. However, through inhibiting the enzyme, hypoxanthine and xanthine accumulate in the body. They are more soluble in water compared with uric acids and are excreted out of the body easily, thus successfully preventing uric acid formation and enhancing purine bases recycling (which are used in DNA and RNA synthesis) [24].

5.2. Treatment

Allopurinol, the most frequently used agent in this class, specifically manages inflammatory diseases, reducing serum urate levels effectively and at the same time leading to decreases in recurrence of gout attacks. Synthesized in the 1950s, allopurinol remains a first-line ULT strategy, proving its reliability and efficacy. Recent research demonstrates that allopurinol also has additional benefits in decreasing cardiovascular disease and hypertension—one study shows a relative risk reduction of 71% in cardiovascular events compared to standard treatments [25]; studies have also suggested a potential positive association between use of allopurinol and improvements in blood pressure and estimated glomerular filtration rate [26]. Moreover, discoveries found allopurinol to be able to slow down the progression of renal disease in patients with chronic kidney disease (CKD) [25]. Therefore, allopurinol is recommended for treating patients with gout who are also faced with high cardiovascular risks or CKD.

Another commonly used xanthine oxidase inhibitor, febuxostat, provides a well-tolerated alternative for patients with allopurinol intolerance or experiencing hypersensitivity. Just like allopurinol, febuxostat lowers uric acid levels through targeting not only xanthine oxidase but also the associated enzyme of xanthine dehydrogenase. This ensures a more reliable inhibition of enzymes, leading to more complete reductions of uric acid [27]. Febuxostat holds many benefits: firstly, febuxostat inhibition of xanthine oxidase will not affect any other purine metabolism activity [28]; secondly, febuxostat is more renoprotective than allopurinol, meaning it prevents the kidneys from being further damaged [29]. Furthermore, febuxostat has a lower risk of interaction with other drugs. A study examined cross interactions of febuxostat with other drugs by analyzing three in vitro systems, finding that febuxostat binding to blood protein is not affected by common drugs like warfarin, febuxostat is metabolized by multiple enzymes, thus inhibition of a single enzyme would not affect overall function, and febuxostat does not inhibit major Cytochrome P450 proteins (CYP) enzymes [30].

5.3. Safety

While allopurinol is generally well-tolerated, adverse effects may occur. Allopurinol is often associated with allopurinol hypersensitivity syndrome, which includes severe skin rash and organ failure . Common side effects are often mild—including skin rash, nausea, and diarrhea . People who took allopurinol also reported problems of gout flares, damage to the kidneys and liver, and blood disorders. Exceedingly rare cases of delayed peripheral neuropathy, Guillain-Barré Syndrome, and leukopenia are also reported . Concerning the safety of the medication, patients need to consider their own physical conditions and drug usage when taking allopurinol. As a purine analogue, allopurinol can interact dangerously with other purine-related drugs like azathioprine. When used without careful management, allopurinol inhibits azathioprine metabolism, leading to toxic azathioprine accumulation. While this combination can be exploited in patients who metabolize azathioprine quickly, the two drugs combined are generally harmful and should be avoided .

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Downsides of using febuxostat as treatment include mild effects of liver function abnormalities, diarrhea, and rash. Amongst the adverse outcomes, cardiovascular events are the most common serious consequences, with the FDA issuing them with a Boxed Warning . Recent searches downgraded febuxostat from first-line to second-line due to the proven strong association between the drug and cardiovascular deaths [28]. Therefore, febuxostat is only reserved for patients who are intolerant to allopurinol.

6. Discussion

After analyzing diverse types of gout treatment, we reached the following conclusions. The drugs that are reviewed prove their effectiveness: NSAIDs are fast-acting and cost-effective, colchicine is highly specific and potent, glucocorticoids offer versatile options, while xanthine oxidase inhibitors prove efficiency in urate lowering. Now we need to consider two pillars—efficacy and safety. Although effective, these drugs hold major problems in side effects: NSAIDs carry GI or CV risks, colchicine has severe toxicity risks and a narrow therapeutic window, glucocorticoids have systemic side effects like gastrointestinal ulcers, while the xanthine oxidase inhibitor febuxostat has an FDA Boxed Warning.

Thus, the central challenge in gout treatment is not a lack of effective drugs—it is rather two key issues: drug-related safety and a dangerous knowledge gap. The latter may be a greater problem. A study conducted in Southern China shows a deficit in understanding: out of 149 primary gout patients and 151 non-rheumatology doctors, only 48.3% of patients and 55.6% of doctors know about optimal serum uric acid, and 29.5% of patients and 43.6% of doctors acknowledge the need for lifelong ULT. Another research reflects how nurses and the partners of patients only know about gout daily rather than in a more in-depth way.

To solve these problems, in the future, researchers can work to synthesize new drugs or to refine current medications to improve the safety profile. Moreover, efforts should be taken to promote gout treatment education to both professionals and patients.

7. Conclusion

This article discusses the mechanism of gout and four current treatments of gout, which are attributed to high blood acid levels, and the article also identifies contributing factors. Regarding the four current treatments, although each treatment has different positive and negative properties, patients should use medications based on individual patient conditions. Moreover, the article shows that there is insufficient understanding of gout among people. Not all doctors and patients fully understand gout, which may lead to the bad effects of the treatment. In conclusion, although people already know the mechanism and some effective treatments, there is still a need for some research about avoiding adverse reactions and new inventions of medications. And to ensure the effectiveness of treatment, it is necessary to provide more comprehensive training for medical staff and disseminate more information about gout to the public.

Authors contribution

All the authors contributed equally, and their names were listed in alphabetical order. Alice is responsible for writing the introduction, current treatment 4 in the main body, and discussion; she is also in charge of revising and editing. Olivia responsible for the abstract and current treatment 1 of

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the main body. Evelyn responsible for current treatment 2 of the main body. Tina responsible for current treatment 3 of the main body and the conclusion.

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