ORIGINAL ARTICLE

Colchicine: The Long Journey of an Ancient Drug to Its Current Uses

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SUMMARY

Colchicine, a natural alkaloid derived from plants of the lilac family, is a substance of ancient use, with more than 2,000 years of history in medicine. Recognized for its anti-inflammatory properties, it acts through multiple mechanisms, which gives it's significant therapeutic value. Currently, it has experienced a renewed clinical interest, being used in various medical specialties, including dermatology.

This article presents a concise overview of the history of colchicine, its main characteristics, action mechanisms, adverse effects and current applications, with special emphasis on its usefulness in the treatment of skin diseases.

INTRODUCTION

Colchicine is an anti-inflammatory and antimitotic agent that has been used for decades in the treatment of various diseases such as gout, familial Mediterranean fever, and acute and recurrent pericarditis. It´s a plant native to Europe, typically found in moist meadows at high altitudes.

In recent years, its use has expanded to a variety of skin diseases, including psoriasis and vasculitis, among other dermatological conditions. There has been ongoing discussion about the significance of this drug's side effects, which may limit its use. Perhaps the most critical of these is overdose, which can result in fatal outcomes. Therefore, colchicine must be used with caution, especially in patients with liver or kidney disease.[1]

ORIGIN OF ITS NAME

The name of this plant derives from the ancient region of Colchis, home to the legendary sorceresses Medea and Circe in Greek mythology. Today, this region corresponds to the Republic of Georgia (Figure 1). According to mythological tradition, the plant was allegedly a favorite of the poisoner Medea, adding a symbolic dimension to its historical narrative.

Across various geographic regions, this plant species has been known by a wide range of popular names, reflecting both its appearance and its perceived properties. Among the most common names are: colchicum, acefrán, wild saffron, meadow saffron, bastard saffron, mysteria, miracle bulb, cholquico, officinal colchicum, dog killer, autumn narcissus, autumn crocus,

Figure 1. The Kingdom of Colchis, shown in green. (Source: Wikipedia).

"quitameriendas" (snack remover), shepherd repellent, shepherd chaser, mountain onion, villorita, and sowing herb, among others.

HISTORY OF COLCHICINE

The medicinal plant known as Colchicum autumnale has a long and fascinating history that dates back to ancient times. As early as 1550 B.C., it was mentioned in the Egyptian Ebers Papyrus as a treatment for rheumatologic diseases, noted for its analgesic and anti-inflammatory properties (Figure 2). Of the approximately 700 medicinal plants described in this compendium, only 18 remain in use in modern medicine, with colchicine being one of the few that has withstood over 40 centuries of medical scrutiny.

In 77 A.D., the Greek physician and botanist Dioscorides distinguished between the less toxic colchicum, used for nutritional, aphrodisiac, and therapeutic purposes, and the wild bulb Colchicum autumnale, which is precisely the species under current study. Likewise, in the writings of Pliny the Elder (23–79 A.D.) and Galen—one of the most influential figures in the history of medicine—colchicine was already recognized as a poisonous substance. Nevertheless, between 129 and 200 A.D., the plant was already being effectively used in the treatment of gout, although its toxic effects at high doses were also well known.

Later, Alexander of Tralles (525–605 A.D.) documented the specific use of colchicine in acute gout episodes, also noting its gastrointestinal side effects, such as gastric and intestinal irritation. Despite these precedents, its use declined markedly in the 16th century due to skepticism from the medical community and Renaissance humanists, who questioned its therapeutic properties and classified it as a poison. In fact, it was even banned by the French School of Medicine between the 16th and 19th centuries.

The resurgence of colchicine occurred in the 18th century thanks to Viennese physician Anton von Störk, who rehabilitated its medicinal use in 1764. Later, in 1783, Husson developed a secret remedy for gout known as Eau Medicinale, whose main ingredient was colchicine. This preparation arrived in the United States in 1798,

Figure 2. he famous Ebers Papyrus.

introduced by Benjamin Franklin, who suffered from gout and experienced significant relief during his stay as ambassador in France.

In the scientific field, pharmacists Pierre-Joseph Pelletier and Joseph Bienaimé Caventou were the first to isolate the alkaloid compound colchicine in 1820. Later, in 1833, Geiger and Hesse managed to purify it further, and in 1884, chemists Laborde and Houdé successfully crystallized the molecule in a reproducible manner. However, its chemical formula was not accurately determined until 1945, when it was finally established by Dewar.

One of the most significant discoveries regarding this substance occurred in 1899, when Sicilian pathologist Biaggio Pernice identified its antimitotic action—a property that has been extensively studied to this day for its potential application in cancer research.[2-4]

CHEMICAL NAME, STRUCTURE, AND CHEMICAL FORMULA OF COLCHICINE[5]

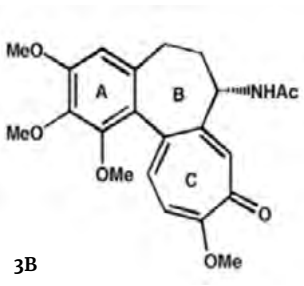
Chemical name: N-[(7S)-5,6,7,9-tetrahydro-1,2,3,10-tetramethoxy-9-oxobenzo[a]heptalen-7-yl]acetamide.

Structure and chemical formula (Figure 3 A–B)
Colchicine and its natural analogues consist of three fused rings with various functional groups (Figure 3B):

- A six-membered aromatic ring with free or alkylated hydroxyl groups (Ring A).
- A cycloheptadiene ring with an acetamido, amino, or alkylamino group (Ring B).
- A seven-membered aromatic ring with carbonyl and methoxy groups, rarely hydroxyl, (Ring C of the tropolone moiety).

Figure 3A: Photograph of the lilaceous plant. Figure 3B: The complex formed by three rings with various chemical components, which bind and undergo modifications that allow colchicine to attach to tubulin.

3A



3B

The presence of a sugar on Ring A gives rise to colchicoside, an analogue of colchicine that is unable to form a complex with tubulin, as occurs when Ring C is replaced in lumicolchicine and colchinol, or when the tropolone structure is modified to produce isocolchicine. However, variations in Ring B do not prevent colchicine from binding to tubulin.[6]

ABSORPTION AND EXCRETION OF COLCHICINE[7]

Colchicine is a substrate of both cytochrome P450 and P-glycoprotein. It is administered orally and primarily absorbed in the jejunum and ileum. In the intestinal epithelium, P-glycoprotein present in enterocytes can secrete unchanged colchicine back into the intestinal lumen, where it may eventually be excreted.

The fraction of the drug that crosses the intestinal barrier enters systemic circulation and is filtered by the renal glomeruli, with partial elimination occurring through the proximal tubules of the kidney. In the liver, colchicine is metabolized into three main metabolites by the enzymatic action of CYP3A4, an isoenzyme of the cytochrome P450 family. This physiologically significant enzyme is predominantly found in the liver and intestine and is encoded by the CYP3A4 gene in humans. Its primary function is to oxidize small foreign organic molecules—such as toxins or drugs (xenobiotics)—to facilitate their elimination from the body.

Both desmethylated metabolites and part of the unchanged colchicine are eventually secreted into the bile by hepatic P-glycoprotein and transported to the duodenum for final intestinal excretion.

FACTORS AFFECTING THE METABOLISM AND EXCRETION OF COLCHICINE

First, it´s important to know that the elimination half-life of colchicine ranges between 26 and 31 hours.[9] Second, that up to 20% of colchicine is excreted in the urine, while the majority enters enterohepatic recirculation to be excreted in bile via the feces.[10] In cases of severe hepatic failure, clearance increases and half-life decreases, contrary to what occurs in severe renal

Figure 4. Diagram illustrating the absorption and excretion of colchicine (Adapted from: Slobodnick et al.[1])

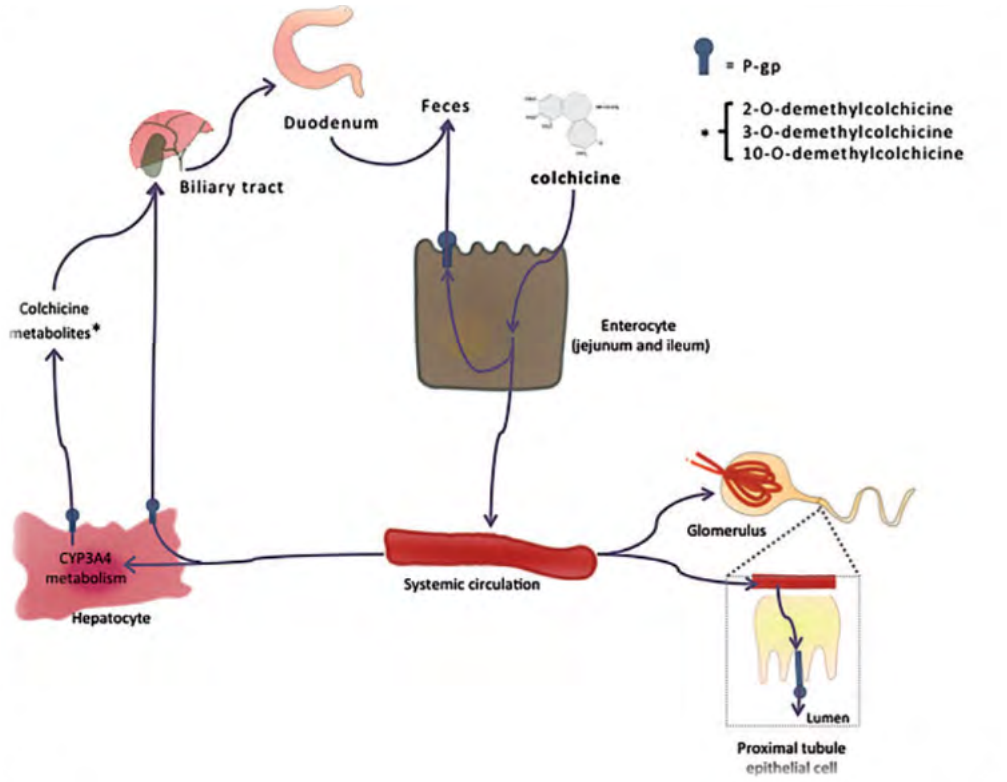
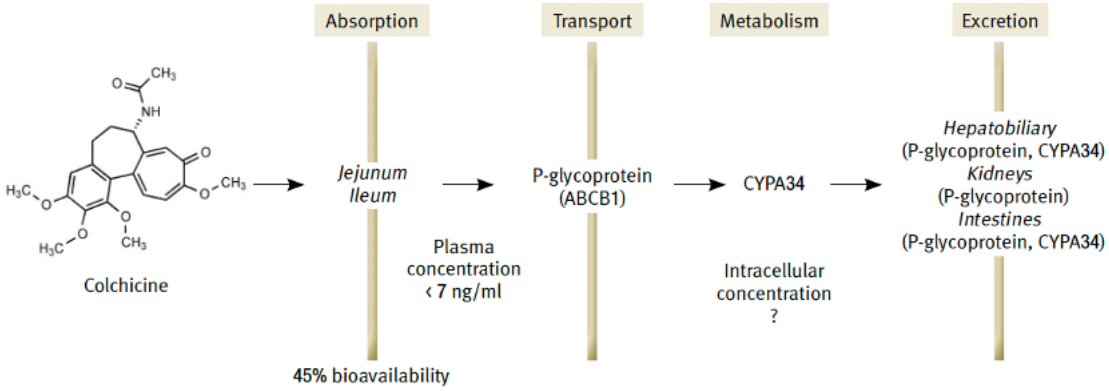


Figure 5. Diagram illustrating the pharmacokinetics of colchicine and the proteins involved in its transport and metabolism (Adapted from: Gül[2])



failure, where clearance decreases by approximately 75%. It is also important to know that colchicine is not eliminated through hemodialysis.

DISTRIBUTION OF COLCHICINE

Oral colchicine reaches a bioavailability of approximately 45%, achieving peak plasma concentration between 24 and 48 hours, with a distribution half-life of 1 to 2.7 hours.[11] In plasma, approximately 40% of colchicine binds to albumin, with high concentrations found in the liver, kidneys, spleen, and gastrointestinal tract.[12]

It is important to highlight that with chronic administration, colchicine can cross the placental barrier. Additionally, colchicine accumulates in leukocytes at levels higher than in plasma and remains within them for several days after oral administration.

MECHANISMS OF ACTION OF COLCHICINE

Its mechanisms of action can be summarized in six categories:

- Action on microtubules
- Action on neutrophils
- Anti-inflammatory action

- Action on macrophages
- Antifibrotic action
- Immunosuppressive action

MECHANISMS OF THE ANTI-INFLAMMATORY ACTION OF COLCHICINE[13]

In summary, this drug exerts multiple effects through its interference with:

- Cell migration
- Cytokine release and intracellular trafficking
- Modulation of inflammatory cell activity
- Alteration of L-selectin expression on neutrophils and redistribution of E-selectin on endothelial cells, thereby inhibiting neutrophil adhesion, extravasation, and recruitment.
- Accumulation in leukocytes, where it interferes with their migration and degranulation, modulating their inflammatory activity.

The diagram provides a general overview of the mechanisms of action of colchicine.

INDICATIONS OF COLCHICINE IN DERMATOLOGY

Although the clinical use of colchicine has a history spanning centuries, its applications have expanded in recent years across various pathologies, with dermatological diseases representing a progressively growing group. The most frequently cited indications (Figure 7) include the following:

LEVELS OF EVIDENCE FOR COLCHICINE IN DERMATOLOGICAL DISEASES PUBLISHED IN 2023[15]

Four levels of evidence were established for these diseases based on the following criteria:

Figure 6. Diagram of the activities of colchicine (Adapted from: Sardana et al.[3])

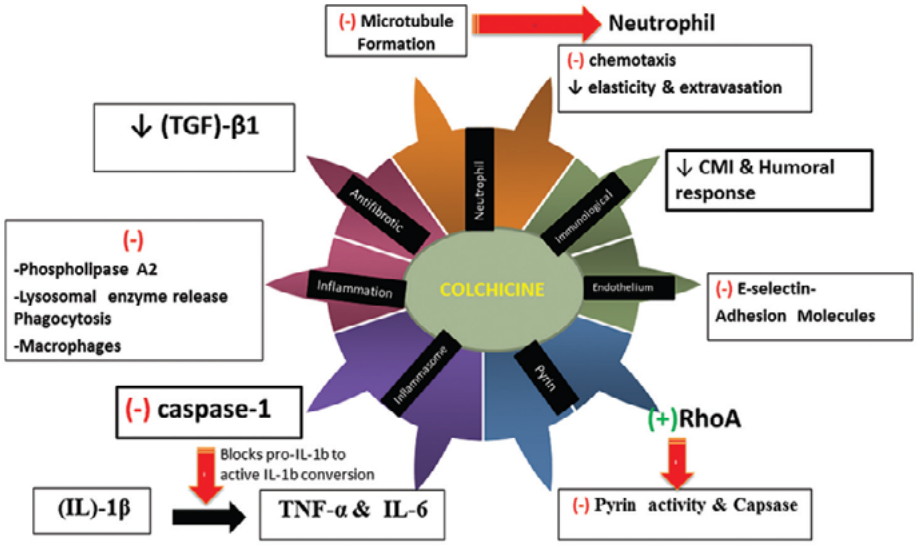
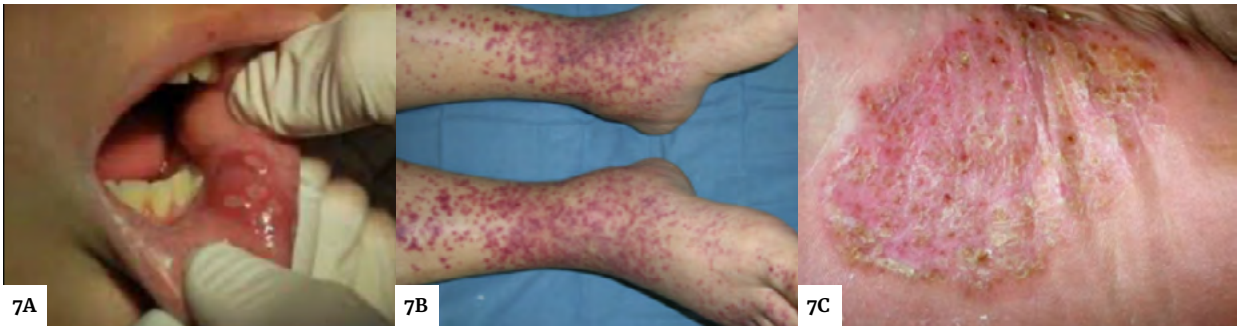


Figure 7A: Recurrent aphthous stomatitis Figure 7B: Leukocytoclastic vasculitis and urticarial vasculitis Figure 7C: Palmoplantar pustulosis.



- **Level I:** At least one randomized controlled trial or meta-analysis.
- **Level II:** At least one controlled study without randomization/quasi-experimental study.
- **Level III:** Non-experimental descriptive studies.
- **Level IV:** Expert group reports/opinions or consensus conferences.

	DISEASE	LEVEL OF EVIDENCE
Neutrophilic Dermatoses	Palmoplantar pustulosis	**Level I**
	Pyoderma gangrenosum	Level IV
	Sweet's syndrome	Level IV
	Recurrent aphthae	**Level I**
Fibrosing Dermatoses	Scleroderma	Level IV
Vasculitis	Leukocytoclastic vasculitis	Level IV
	Behçet's disease	Level II
Precancerous Dermatoses	Actinic keratoses	**Level I**

Table 1. Graphical representation of the main dermatoses evaluated according to the level of evidence (Adapted from: Huber et al.[4])

RESULTS OF COLCHICINE TREATMENT IN VARIOUS DERMATOLOGIC DISEASES

Given the numerous reports on the use of colchicine in dermatologic conditions, a literature review was conducted, and the findings are presented below.

Several dermatologic diseases have recently been evaluated regarding the efficacy of colchicine treatment. In 2023, data were published classifying these conditions according to levels of scientific evidence, providing a clearer overview of its potential therapeutic indications and clinical support. Below is a summary of the main dermatoses evaluated:

Palmoplantar pustulosis: Its efficacy has been known for over 30 years. The dose ranges from 0.5 to 1–2 mg/day, divided into three doses, with results ranging from very good to moderate. However, side effects can be significant, according to a Cochrane review published in 2006.[16]

Pyoderma gangrenosum: Individual reports and successful case series using 2–4 mg/day for 2–4 months. Parren et al. reported a case of penile pyoderma gangrenosum successfully treated with low doses of colchicine.[17]

Sweet's syndrome: Colchicine has been used as a steroid-sparing agent. A retrospective analysis in 20 patients showed a 90% improvement with 1–1.5 mg/day for 10–21 days, including improvements in skin lesions, fever, and arthralgia.[18]

Recurrent aphthous stomatitis: A study by Cabras et al. in 2020 reported that colchicine yielded similar results to prednisone at 5 mg daily for 12 weeks, though inferior to clofazimine, thalidomide, or dapsone.[19]

Scleroderma: Reports describe improved skin elasticity, mouth opening, and finger mobility with 1.2 mg/day over approximately 40 months of treatment.[20]

Small- and medium-vessel vasculitis: To date, studies have not shown significant results.

Behçet's disease: A study in Turkey involving 116 patients administered 1–2 mg/day of colchicine, resulting in a significant reduction of genital but not oral lesions—this effect was observed only in female patients.[21]

Hypertrophic actinic keratosis: A 1% colchicine cream applied twice daily for 10 days showed lesion reduction after 60 days of therapy, with no relapse during two months of follow-up.[22]

Psoriasis: Topical 1% colchicine or systemic administration (0.02 mg/kg/day) showed good results only in mild cases. Reports exist of successful combination therapy with biologics in patients unresponsive to monotherapy.[15,23]

Neutrophilic eccrine hidradenitis: Only short-term reports show good results.

Hidradenitis suppurativa: No results with monotherapy. Good outcomes reported when combined with minocycline (100 mg/day + 0.5 mg colchicine BID for 3 months) or doxycycline (100 mg/day + 1 mg colchicine/day), improving both clinical condition and quality of life.[1,24]

Acne vulgaris and acne fulminans: 1 mg/day for 2 months led to 70% improvement in nodular/cystic inflammatory acne, considered a third-line agent. In acne fulminans unresponsive to isotretinoin, adding colchicine at 1 mg/day for 2 months yielded excellent results, particularly in a report by Durdu involving 12 patients—though gastrointestinal side effects were noted.[1,25]

Chronic urticaria: Few studies to date. Partial or complete response reported, but use remains controversial. Nobuya Abe et al. described a case of spontaneous chronic urticaria resistant to antihistamines and dupilumab, treated with 0.5 mg colchicine twice daily with surprising lesion resolution.[26]

Erythema elevatum diutinum: Isolated reports of short cycles with 0.5 mg BID achieving success.

Erythema nodosum: A report on 12 patients refractory to hydroxychloroquine, dapsone, and potassium iodide showed very good response to colchicine over 2–3 months; in two cases, colchicine was combined with dapsone in one patient and prednisone in another.[27]

Cutaneous amyloidosis (macular and papular forms): 0.5 mg BID for 3 months improved pruritus and papules. Reports are scarce and contradictory.

Cutaneous sarcoidosis: Systemic colchicine plus topical corticosteroids showed good results in three cases.

Other isolated case reports include Facial granuloma Excellent response to colchicine in a case reported by Ya Thang Yang.[28] Generalized granuloma annulare: Greensait et al. presented a study of 12 cases comparing colchicine, minocycline, and pentoxifylline. While hydroxychloroquine is classically preferred, colchicine was nearly as effective with a shorter treatment duration.

Acquired perforating dermatosis A rare condition primarily treated with prednisone. Kharghoria[30] et al. reported a case where colchicine at 1 g daily led to excellent results after failed oral steroid treatment.

Acute generalized exanthematous pustulosis: A case raised the possibility of colchicine as a treatment option after favorable results in a patient with a rash on day five of hydroxychloroquine use for COVID.[31]

OTHER SPORADIC REPORTS OF COLCHICINE USE IN VARIOUS DERMATOSES INCLUDE[22]

- Granulomatous pigmented purpuric dermatosis: A single case unresponsive to other therapies achieved remission in 3 months with 1 mg/day of colchicine.
- Linear IgA bullous dermatosis: Successful treatment reported in both children and adults with doses ranging from 0.5 to 1.5 mg/day.
- IgA pemphigus and pemphigus foliaceus: Isolated reports of successful treatment with colchicine at 1.5 mg/day.
- Bullous pemphigoid: Recommended as a maintenance therapy in a study involving 15 patients.
- Epidermolysis bullosa: Various clinical forms treated successfully with doses between 0.5 and 2 mg/day.
- Dermatitis herpetiformis: A single report of treatment with 0.6 mg four times daily; however, diarrhea was noted as a complication.

ROUTES OF ADMINISTRATION OF COLCHICINE[32]

Colchicine is primarily administered orally, available in tablet or drop formulations. Intravenous administration is indicated only in situations requiring a rapid therapeutic response; however, this route carries a higher risk of systemic toxicity due to the absence of first-pass metabolism and gastrointestinal symptoms that could otherwise serve as early warnings of overdose.

Topical formulations have also been developed, such as a 1% hydrophilic gel and creams with concentrations ranging from 0.5% to 1%, typically applied twice daily (BID).

DOSAGE RECOMMENDATIONS FOR COLCHICINE[32]

It is recommended to use low, divided doses to improve gastrointestinal tolerance. Once-daily (QD) dosing can help enhance treatment adherence. Dose adjustments are essential in patients with moderate to severe renal insufficiency and in elderly individuals. Potential drug interactions should also be closely monitored.Colchicine use is contraindicated in patients with creatinine clearance below 10 ml/min and in those with severe hepatobiliary dysfunction.

OPTIMAL DOSAGE IN DERMATOLOGY

In dermatologic literature, most studies recommend a daily colchicine dose ranging from 0.5 mg to 2 mg. This can be administered once (QD), twice (BID), or three times (TID) per day, depending on the clinical case and the patient's individual tolerance. Prolonged use is considered safe, supported by its long-term administration in patients with familial Mediterranean fever, who receive it throughout their lives.

DRUG INTERACTIONS WITH COLCHICINE

The following table presents medications that are strong and moderate inhibitors of cytochrome P450, as well as P-glycoprotein inhibitors.[7]

Among the medications that can increase plasma colchicine concentration are various commonly used drugs that interfere with its metabolism.

Among antibiotics, clarithromycin and erythromycin are notable; among antifungals, fluconazole, itraconazole, and ketoconazole are the main ones implicated. Similarly, calcium channel blockers such as diltiazem and verapamil can also enhance its effect. In the group of immunosuppressants, caution should be exercised with cyclosporine and tacrolimus.

Finally, in the statin group, both atorvastatin and simvastatin may increase the risk of colchicine toxicity due to pharmacological interactions.

Table 2. Medications grouped according to the type of inhibition that can increase plasma colchicine concentration (Taken from: Robinson et al[5])

STRONG INHIBITORS OF CYTOCHROME P450	STRONG INHIBITORS OF CYTOCHROME P450	P-GLYCOPROTEIN INHIBITORS
Claritromicina	Cimetidina	Amiodarona
Cobicistat	Ciprofloxacino	Carvedilol
Diltiazem	Ciclosporina	Claritromicina
Itraconazol	Eritromicina	Itraconazol
Ketoconazol	Fluconazol	Quinidina
Ritonavir	Fluvoxamina	Ranolazina
Telitromicina	Imatinib	Ritonavir
Voriconazol	Verapamilo	Verapamilo

ADVERSE EFFECTS AND TOXICITY OF COLCHICINE[1]

The most common side effects of colchicine occur at the gastrointestinal level, including nausea, vomiting, and diarrhea, which affect approximately 5% to 10% of treated patients. Less frequent adverse reactions include elevated liver transaminases, myotoxicity, and alopecia. It is important to note that the dose required to cause morbidity or mortality can vary among individuals and usually manifests clinically within the first 24 hours after ingestion, with the onset of generalized organ dysfunction.

COLCHICINE TOXICITY[33]

Between 1968 and 2021, only four cases of dermatological manifestations attributed to colchicine toxicity have been reported. The predominant lesions were blistering in nature, including conditions such as toxic epidermal necrolysis, erythema multiforme, and Sweet's syndrome. One isolated case of alopecia was also documented. It is noteworthy that all cases involved drug overdose, and in some, concomitant ingestion of allopurinol was identified. The small number of reports suggests that these dermatologic complications are rare and infrequently observed in routine clinical practice.

PHASES OF COLCHICINE INTOXICATION[34]

Three phases have been reported, based on the time elapsed since colchicine ingestion: Phase 1 (0 to 24 hours): Gastrointestinal phase. Phase 2 (1 to 7 days): Characterized by multiorgan failure. Phase 3 (more than

7 days): Considered the recovery or death phase. This table outlines the clinical manifestations associated with each phase.

Table 3. Phases of Colchicine Poisoning.

Phase I (0–24h): Gastrointestinal	Nausea, vomiting, abdominal pain, bloody diarrhea, dehydration, and leukocytosis. Shock may occur due to hypovolemia and cardiac insufficiency.
Phase II (1–7 days): Multiorgan failure	Cardiac arrhythmias, liver failure, seizures, pancytopenia, metabolic changes, neurological syndrome with abnormalities in proximal extremities, distal sensory abnormalities, and altered neural conduction.
Phase III (+7 days): Recovery or death	Alopecia, myopathy, neuropathy, mononeuropathy, or rebound leukocytosis; death is usually caused by respiratory failure, intractable shock, arrhythmias, and cardiovascular collapse.

CONTRAINDICATIONS OF COLCHICINE35

The use of colchicine requires careful prior clinical evaluation due to its potential risks in certain medical conditions. Below are the main contraindications, grouped by clinical category.

Table 4. Contraindications of Colchicine

CATEGORY	SPECIFIC CONTRAINDICATION
Allergy or hypersensitivity	Hypersensitivity to the active ingredient or excipients
Physiological conditions	Pregnancy
Severe organ dysfunction	Severe hepatic or renal insufficiency; patients undergoing hemodialysis
Digestive disorders	Severe gastrointestinal disorders or gastric ulcer
Cardiovascular disorders	Cardiac abnormalities
Hematological disorders	Blood dyscrasias
Recent drug interactions	Use of CYP3A4 and/or P-glycoprotein inhibitors within the past 14 days

It is essential to consider these contraindications before initiating colchicine treatment, as overlooking them may lead to serious adverse events, especially in patients with organ dysfunction or those undergoing treatment with drugs that alter its metabolism.

CONCLUSION

Colchicine is one of the oldest drugs in the history of medicine, although its mechanism of action is still not fully understood. Currently, its use in dermatology has gained relevance as a second- or third-line therapeutic option in various skin conditions.

Despite its well-known toxicity at high doses, it is considered safe and well tolerated at appropriate doses. Its topical application has shown effectiveness in conditions such as plaque psoriasis and actinic keratosis.

However, much of the available evidence comes from anecdotal reports and small case series, highlighting the need for more robust studies to more clearly establish its role in modern dermatology.

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