Journal for Research in Applied Sciences and Biotechnology

www.jrasb.com

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

Review on Medicinal Herbs in the Treatment of Gout

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www.jrasb.com || Vol. 3 No. 6 (2024): December Issue

Received: 21-11-2024	**Revised:** 27-11-2024	**Accepted:** 04-12-2024

ABSTRACT

Joint urate crystals are the hallmark of the inflammatory condition known as gout. Acute gout is characterised by the abrupt development of swelling and pain that begins in the greater toe. This condition is linked to high levels of uric acid in the blood, which can occur when the kidneys do not excrete enough of the acid or when the body produces too much uric acid. Allopurinol, which inhibits the enzyme xanthine oxidase and hence lowers blood uric acid levels, is the mainstay of conventional gout treatment in contemporary medicine. Pain and inflammation can be managed with the help of nonsteroidal anti-inflammatory drugs (NSAIDs). It has also been seen that non-pharmacological approaches, such as dietary improvements, can improve patients' situations. Many people believe that herbal treatments are safer and more effective than conventional pharmaceuticals, especially when it comes to patient adherence and the potential for adverse effects. Celery and parsley, when used together, have demonstrated effects comparable to those of allopurinol. This article's goal is to provide a summary of allopurinol's herbal substitutes.

Keywords- inflammation, hyperuricemia, allopurinol, monosodium urate crystal, xanthine oxidase inhibitor**.**

I. INTRODUCTION

Gout has been widely acknowledged as a medical ailment since the time of Hippocrates. Recent advances in our understanding of the disease's biology and a small change in its distribution pattern have kept the disease in the medical literature. Gout is the result of a cascade of physiological irregularities that lead to the deposition of uric acid ions and crystals in and around joints and soft tissues. Decreased renal clearance of uric acid is the main cause of gout.[1] Acute gout, which affects a large percentage of American adults, is an inflammatory arthritis characterised by periodic, harmless attacks of synovitis caused by the buildup of crystals of monosodium urate. Obesity, related comorbidities, longevity, and iatrogenic causes of hyperuricemia, such as diuretics, are associated with an increasing prevalence of gout, according to epidemiologic studies. The symptoms of an acute flare-up of gouty arthritis include a decrease in joint range of motion, swelling, redness, heat, and pain all occurring suddenly. In the absence of treatment, flare-ups can last for hours or weeks, causing the affected individual to miss work and potentially progressing to a chronic condition that debilitates their joints. When the causes of acute gout episodes are left untreated, the frequency of flare-ups increases with time.[2]

In Chronic gout is a degenerative urate metabolism disease that causes inflammation and the buildup of monosodium urate crystals in soft tissues and joints. It is twice as common in men as in women, and it is the most common form of inflammatory arthritis in men over the age of 40.the third Joint structures can be damaged by long-term hyperuricemia, which is a

Journal for Research in Applied Sciences and Biotechnology

www.jrasb.com

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

symptom of chronic gouty arthritis, chronic gout, or both. This condition is usually associated with the development of tophi, which are subcutaneous MSU deposits. In the end, gout becomes more difficult to treat and more severe over time, eventually developing into "refractory gout" as deposits continue to build up.[3,4] Any systemic illness that increases the likelihood of hyperuricemia can increase the risk of acquiring gout symptoms. Modifiable risk factors include things like alcohol consumption, obesity, a diet heavy in purines, and diuretic medication. According to studies, gout is more common among people who eat red meat and seafood, but dairy products might help keep the disease at bay.the three, five The main cause of gout, according to most people, is hyperuricemia. Hyperuricemia is defined as a rise in blood serum uric acid (UA) levels above 6.8 mg/dl, which causes MSU crystallisation because the solubility threshold has been exceeded.

Extremely common, with a prevalence of around 20%, is hyperuricemia. But, in the span of 10–15 years, fewer than half of those whose Uric Acid levels are 10 mg/dl will suffer from gout. new research has shown. Although the epidemiology varies among ethnic groups, the incidence of gout is around 1-3 per thousand person-years. Approximately 4% to 6% of the global population is affected by this illness. The majority of the world's population suffers from gout, an inflammatory arthritis. Epidemiological evidence shows that gout's incidence and prevalence have been on the rise in recent years. The incidence of gout has skyrocketed in the last two decades across Europe and the United States. In the UK, almost 700,000 people suffer with gout. Hospitals in the United Kingdom see an estimated four million cases of outdoor gout annually. Gouty arthritis affects an estimated 1.4 females and 4 males per thousand patients annually, according to another study. [6, 7]

There are two main mechanisms that lead to hyperuricemia and the production of MSU crystals: an increase in purine synthesis and a decrease in UA excretion. Once the monosodium urate solubility threshold is exceeded, crystals of MSU will develop. Haematologic cancers, metabolic syndrome, psoriasis, cytotoxic drugs and chemicals, and alcohol are among the many potential causes of purine overproduction.[8] Diabetes, high blood pressure, stroke, and myocardial infarction are the most common comorbidities associated with gout. When additional health problems are present alongside gout, the overall life expectancy drops and the mortality rate climbs. Recent research has demonstrated that gout causes or exacerbates a number of serious cardiovascular diseases.[9] Due to factors such as inadequate disease care, inaccurate diagnosis, a lack of knowledge about diseases and their treatments, a communication gap between patients and healthcare providers, and an inability to obtain necessary medications, the worldwide economic burden of disease has grown substantially. [10]

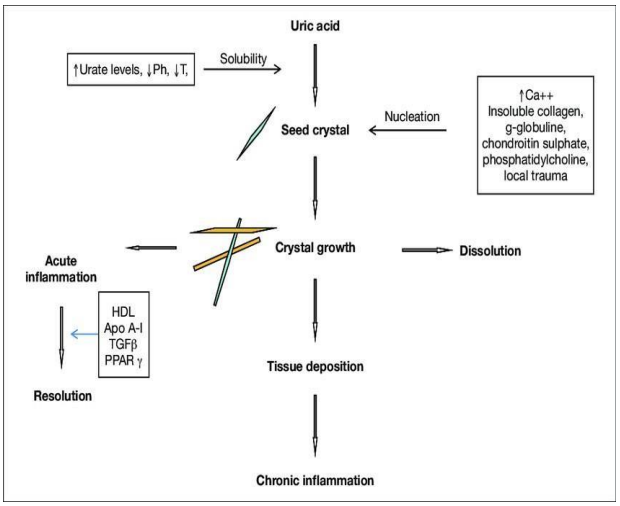

Fig: 1 The pathophysiology of monosodium urate crystal formation can be illustrated in the following flowchart

II. CONVENTIONAL METHOD FOR MANAGEMENT OF GOUT XANTHINE OXIDASE INHIBITORS

Inhibitors of xanthine oxidase (XO) are substances that prevent the enzyme from doing its job. Two important reactions that XO catalyses are the formation of hypoxanthine from xanthine and the transformation of hypoxanthine to uric acid. That is why blocking XO reduces the production of uric acid and its precursor. Both febuxostat and allopurinol reduce SU concentrations by blocking XO. The main medication in this class used to treat persistent gout is allopurinol. Another urate-lowering medication, febuxostat, has recently received approval for dosages ranging from 40 to 80 mg in the United States and 80 to 120 mg in Europe. Allopurinol and this non-purine xanthine oxidase inhibitor are considered to be equally effective.[11-14.]

Uricosurics:-

Medications that promote the renal excretion of uric acid, known as uricosurics, are helpful in the management of SU. Substances that obstruct the kidney's organic anion transporters (OATs) include probenecid and benzbromarone. These transporters are responsible for facilitating the transfer of uric acid from the urine to the blood. Patients with a preexisting condition of kidney stones should be cautious when using uricosurics due to the possibility of developing uric acid stones. [15-16].

Uricase :-

Uric acid (UA) is a result of purine metabolism. The enzyme uricase, sometimes called urate oxidase, converts UA into allantoin, a molecule that is very soluble in water. The enzyme uricase plays a crucial role in the body's oxidoreductase system. Maintaining a steady level of uric acid in the body is dependent on this

Journal for Research in Applied Sciences and Biotechnology

www.jrasb.com

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

enzymatic process. However, hyperuricaemia has developed because evolution has silenced the gene that encodes uricase, which has led to the accumulation of uric acid in humans and other primates.Citations [17]

Table 1: Following table represents the urate lowering methods and also the control of inflammation in recent therapies.

Urate lowering drugs	Anti-inflammatories
A. Xanthine Febuxostat (40-120mg/day) **B. Uricosurics** Probenecid (250mg-1g/day) Benzbromarone (50- 200mg/day) **C. Uricases** Pegloticase (8mg i.v every 2 week)	**A.** Colchicine (1.2 mg followed by 0.6mg in1 h) **B. NSAIDs** Indomethacin (50mg three times a day) Ibuprofen and paracetamol tablet **C. Glucocorticoids** Prednisolone

Patients fear long-term negative effects and hence do not adhere to urate reduction treatments. Stevens- Johnson syndrome, hypersensitivity syndrome, renal toxicity, and fatal liver necrosis are some of the adverse effects of allopurinol, a commonly used XO inhibitor. [18] The kidney-harming effects of selective COX-2 inhibitors are similar to those of non-selective NSAIDs, but they are less severe. Nephrotic damage, gastrointestinal issues, and deadly hypersensitivity syndrome have been associated with urate-lowering drugs such XO inhibitors and uricosuric agents. Nausea, vomiting, and severe diarrhoea are common side effects of colchicine. Although cytokine inhibitors are highly successful with few adverse effects, they are relatively expensive when compared to more traditional therapy options. The 20 participants (75 percent men) in the UK gout trial who had ULT also reported frequent gout flare-ups, in addition to other side effects. [19] On top of that, several patients expressed concern about the potential side effects of their drug and were thus hesitant to take it for the rest of their lives.[20,21]

Role of medicinal plants as xanthine oxidase inhibitors

Since plant-based medications are thought to be far safer than synthetic ones, their usage in treating a variety of illnesses is growing globally. India is a true and abundant storehouse of aromatic and medicinal plants. A total of 4,000 of the more than 17,500 wild plant species found in India have therapeutic uses. The market for herbal goods is expanding quickly, as are research efforts. Due to its natural nature and documented long history of usage as folk medicine, herbal medicines are generally considered safe and non-toxic when compared to allopathic medications.[22]

A wide variety of these plants are used in the treatment of gout. Some plants can mimic the mechanism of action of allopurinol as a xanthine oxidase inhibitor.[23]

Table 2: plants which can help in the treatment of gout

Scientific name	Family	Common name	Part used
Apium graveolens	Apiaceae	Celery	Seed
Petroselinum crispum	Apiaceae	Parsley	Seed
Dendrobium candidum	Orchidaceae	Noble Dendrobium	Leaf
Asparagus racemosus	Asparagaceae	Shatavari	Roots
Brassica oleracea	Brassicaceae	Cabbage	Leaves
Cannabis sativa	Cannabaceae	Bhang	Leaf
Zingiber officinale	Zingiberaceae	Ginger	Rhizomes
Prunus avium	Rosaceae	Sweet cherry	Fruit
Paullinia pinnata	Sapindaceae	Climbing shrub	Leaf
Coffea arabica	Rubiaceae	Coffee	Beans
Withania somnifera	Solanaceae	Ashwagandha	Root and stem
Caryophyllus aromaticus	Myrtaceae	Clove	Flower buds
Curcuma longa	Zingiberaceae	Turmeric	Whole plant

Fig: 2 Herbal plant benefit as gout Apium graveolens

Celery has shown promise as a herbal remedy for gout and might be suggested as a natural alternative to synthetic medications like allopurinol. Scientific proof is still needed, though, before these natural medications can be used. The current study showed that celery seed extract (CSE) significantly reduced the serum uric acid

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

level. [24,25] This is consistent with a 2018 study by Dolatti et al., which discovered that an extract from A. graveolens can reduce serum uric acid levels by inhibiting hepatic xanthine oxidase activity. It has also demonstrated practical use as a useful bioactive agent or functional food that can cause hyperuricemia. The inhibition of xanthine oxidase activity is most likely how CSE works.[26]

It was observed in a study that oral administration of celery, leek and parsley decreased level of uric acid level and creatinine in gouty rats, which is consistent with this study.

In a recent study, Shaopeng et al. (2019) found that both the oil and aqueous extracts of celery seeds reduce edema in the ankle joints of rats with gouty arthritis and lower the levels of uric acid in the serum of mice with hyperuricemia.[27]

Table 3: Effect of different doses of celery seed extract on gout compared to allopurinol

Groups	Zero mean±SD (mg/dl)	1st (mg/dl)	3rd (mg/dl)	7th (mg/dl)
Group Control group)	3.3±0.28	2.9±0.24	3.5±0.34	3.6±0.28
Group	3.2 ±0.24	3.4 ±028	6.5 ±0.21	9.0±0.45
Group C (CSE 100 mg/kg)	3.4 ±0.46	3.5 ±0.51	4.5 ±0.38	4.6 ±0.25
Group D (CSE 200 mg/kg)	3.6 ±0.34	3.7 ±0.44	3.5 ±0.34	3.3±0.43
Group E (CSE 400 mg/kg)	3.3 ±0.49	3.5 ±0.38	3.4 ±0.30	3.0 ±0.25
Group (Allopurinol)	3.0 ±0.34	3.4 ±0.48	3.1±0.42	2.9±0.38

There is no significant side effects are seen in the celery seed extract and hence itcan be potentially considered for the treatment of gout.[28]

Petroselinum crispum

Aqueous parsley leaf extract's impact on mice with oxonate-induced hyperuricemia was shown in an in vivo study using a number of methods. Also, it acts against oxidative stress that occurs in hyperuricemic mice by: lowering the level of interleukin 1 beta (IL-1β) and tumour necrosis factor alpha (TNF-α).[29]

The patients with hyperuricaemia are also commonly diagnosed with the urolithiasis and parsley is seen to have a positive effect in alleviating this condition.[30]

A research was performed on rats to note the effect of parsley and celery in gout and also on liver and kidney functions. Also, their combination was injected to see their synergistic effects.[31]

Table 4: Ameliorative effects of parsley and celery on serum kidney and liver biomarkers in oxonate induced hyperuricemia

	Creatinine (mg/dl)	BUN (mg/dl)	Uric acid (U/l)
Control	0.67 ± 0.05	11.1 ± 1	4.5 ± 0.2
HU	1.6 ± 0.15	26.3 ± 1.3	15.7 ± 0.9
HU + ALP	0.7 ± 0.05	14.9 ± 0.8	8.01 ± 0.4
Parsley	0.55 ± 0.01	12.6 ± 1.6	5.8 ± 0.6
Celery	0.6 ± 0.02	12.9 ± 1.5	5.7 ± 0.3
HU + Parsley	0.6 ± 0.02	14.5 ± 1.13	7.8 ± 0.4
HU + Celery	0.6 ± 0.03	13.5 ± 1.8	6.9 ± 0.3
HU + Par + CEL	0.5 ± 0.07	10.4 ± 0.9	5.3 ± 0.2

Values are means ± standard error (SEM) for 7 different mice per each treatment. ALP: Allopurinol; PAR: parsley; CEL celery; HU: hyperuricemia.

A significant improvement in the creatinine, BUN, Uric acid was seen in the mice who were injected with the combination of parsley and celery. A synergistic effect is seen when combination of parsley and celery is given and it improves the kidney functions. They help in the removal of uric acid from the blood.[32]

Table 5: Ameliorative effects of parsley and celery on serum xanthine oxidase and hepatic xanthine oxidase.

	Serum XO (U/l)	Hepatic XO (U/g tissue protein)
Control	13.9 ± 1.1	17.3 ± 1.2
HU	55.7 ± 4	67.2 ± 6.8
HU + ALP	24.4 ± 2.5	27.1 ± 1.7
Parsley	14.9 ± 1.3	17.5 ± 1.9
Celery	14.4 ± 2.9	15.4 ± 1.1
HU + Parsley	23.1 ± 4.7	25.4 ± 2.4
HU + Celery	22.2 ± 4.8	29 ± 4.3
HU + Par + CEL	17.8 ± 3.2	20.1 ± 1.9

Ameliorative effects of parsley and celery on serum and liver xanthine oxidase activity in hyperuricemic mice. ALP: Allopurinol; PAR: parsley; CEL celery; HU: hyperuricemia; XO: xanthine oxidase.[33]

The levels of serum xanthine oxidase and hepatic xanthine oxidase dropped when mice were injected with the combination of parsley and celery

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

together. There effects were similar to the effects of allopurinol. Along with the removal of uric acid they also inhibit the xanthine oxidase in serum as well as in hepatic cells.[34]

Dendrobium candidum

Dendrobium candidum, a traditional Chinese medicinal herb, is believed to have anti- inflammatory and antioxidant properties that may benefit individuals with gout. Its active compounds can help reduce uric acid levels, potentially alleviating gout symptoms such as joint pain and swelling. Additionally, Dendrobium candidum may support overall kidney function, aiding in the elimination of uric acid from the body. While preliminary studies and anecdotal evidence suggest its effectiveness, more rigorous clinical trials are needed to confirm these benefits and establish optimal dosages for managing gout effectively.[35]

Asparagus racemosus

Asparagus racemosus, commonly known as Shatavari, is an adaptogenic herb traditionally used in Ayurvedic medicine. It is known for its potential effects on reproductive health, particularly for women. Shatavari may help regulate hormonal balance, support menstrual health, and enhance fertility. Its adaptogenic properties can reduce stress and promote overall well-being. [36] Additionally, it is believed to have antioxidant and anti- inflammatory effects, supporting immune function and digestive health. While promising, more research is needed to fully understand its mechanisms and efficacy.[37]

Brassica oleracea

Brassica oleracea, commonly known as cabbage, may have beneficial effects for managing gout due to its anti-inflammatory and antioxidant properties. Rich in vitamins C and K, as well as fiber, cabbage can support overall health and may help reduce uric acid levels in the body, which is crucial for gout management. Its low purine content makes it a suitable food choice for individuals with gout, as high-purine foods can trigger flare-ups. Incorporating Brassica oleracea into a balanced diet may contribute to alleviating symptoms and promoting joint health, though further research is warranted to confirm these effects.[38]

Cannabis sativa

Cannabis sativa may offer potential relief for gout symptoms due to its anti- inflammatory properties. Compounds like cannabidiol (CBD) and tetrahydrocannabinol (THC) interact with the body's endocannabinoid system, which can modulate pain and inflammation. Some studies suggest that cannabis may help alleviate acute pain during gout attacks and improve overall quality of life for sufferers. However, while it may provide symptomatic relief, it does not directly lower uric acid levels, which is essential for long-term gout management.[39]

Withania somnifera

Withania somnifera, commonly known as ashwagandha, may have beneficial effects for managing gout due to its anti-inflammatory and antioxidant properties. It helps reduce oxidative stress and inflammation, which are key factors in gout attacks. Some studies suggest that ashwagandha may support overall joint health and potentially lower uric acid levels, though more research is needed to establish these effects specifically for gout. Additionally, its adaptogenic qualities can help manage stress, which may indirectly benefit those with gout.[40]

Caryophyllus aromaticus

Caryophyllus aromaticus, commonly known as clove, may offer potential benefits for managing gout due to its anti-inflammatory and antioxidant properties. Clove contains compounds like eugenol, which has been shown to reduce inflammation and pain, potentially alleviating symptoms during gout flare-ups. Its antioxidant effects may also help combat oxidative stress associated with gout. Additionally, clove has a low purine content, making it a safe spice for individuals with gout. While incorporating cloves into the diet may provide relief, further research is needed to fully understand their effectiveness in gout management.[41]

Curcuma longa

Curcuma longa, commonly known as turmeric, is renowned for its anti- inflammatory properties, primarily due to its active compound, curcumin. This herb may help alleviate gout symptoms by reducing inflammation and pain associated with flare-ups. Curcumin has been shown to inhibit inflammatory pathways, potentially lowering uric acid levels in the body, which is crucial for gout management.[42] Additionally, its antioxidant properties may combat oxidative stress linked to joint damage. Incorporating turmeric into the diet may provide supportive benefits for individuals with gout, but further research is necessary to confirm these effects.[43]

Zingiber officinale

Zingiber officinale, commonly known as ginger, may offer therapeutic benefits for managing gout due to its anti-inflammatory and analgesic properties.[44] Ginger contains compounds like gingerol, which can help reduce inflammation and pain associated with gout attacks. Some studies suggest that ginger may also aid in lowering uric acid levels, which is crucial for preventing flare-ups. Additionally, its antioxidant properties can help combat oxidative stress, further supporting joint health. Incorporating ginger into the diet, whether fresh, powdered, or as a tea, may provide relief for individuals with gout. [45]

Prunus avium

Prunus avium, commonly known as sweet cherry, may have beneficial effects for managing gout due to its anti-inflammatory properties and ability to lower uric acid levels. Studies suggest that consuming sweet cherries can help reduce the frequency of gout

ISSN: 2583-4053
Volume-3 Issue-6 || December 2024 || PP. 33-40
https://doi.org/10.55544/jrasb.3.6.5

attacks and alleviate symptoms.[46] The presence of antioxidants, particularly anthocyanins, in sweet cherries may contribute to their anti- inflammatory effects, helping to reduce joint pain and swelling during flare-ups. Including sweet cherries in the diet can be a tasty and effective strategy for gout management.[47-50]

Paullinia pinnata

Paullinia pinnata, commonly known as the sacha inchi or mountain peanut, has shown potential benefits for gout management.[51-55] Its anti-inflammatory properties may help reduce the swelling and pain associated with gout attacks. The plant contains compounds that can lower uric acid levels, which is crucial for preventing gout flare-ups. Additionally, its rich antioxidant content may protect against oxidative stress, further aiding in joint health. While traditional uses[56] suggest beneficial effects, more clinical research is needed to fully establish its efficacy and safety in gout treatment.[57]

Coffea arabica

Coffea arabica, or Arabica coffee, may have a positive impact on gout management. Some studies suggest that moderate coffee consumption can lower serum uric acid levels, potentially reducing the risk of gout attacks.[58] The antioxidants and anti-inflammatory compounds in coffee may help mitigate inflammation associated with gout. However, individual responses can vary, and excessive caffeine intake may lead to dehydration, which can exacerbate gout symptoms. It's essential for individuals to monitor their coffee consumption.[59-60]

III. CONCLUSION

Gout treatment with medicinal plants is a fascinating and developing field of study. Numerous plants, such as sweet cherry, ginger, turmeric, celery, and parsley, have the ability to act as natural xanthine oxidase inhibitors, providing gout treatment alternatives or supplements. More thorough clinical research is required to determine the efficacy and safety of certain plants, even if they may help reduce uric acid levels and provide symptom alleviation. For individuals who suffer side effects from traditional drugs, the potential of these plants presents a viable avenue for the creation of safer and more easily accessible gout remedies.

REFERENCES

[1] Monu Johnny U.V., Pope Thomas L, Gout: a clinical and radiology review, Radiologic Clinics of North America 2004, Page No- 169-184.

[2] Khanna Puja P, Gladue Heather S, Singh Manjit K, Fitzgerald Jond D, Bae Sangmee, Prakash Shraddha, Kaldas Marian, Gogia Maneesh, Berrocal Veronica, Townsend Whitney, Terkeltaub Robert, Khanna Dinesh,Treatment of acute gout: A systematic review, Seminars in Arthritis and Rheumatism 2014 Page No-31-38.

[3] Brook Richard A, Forsythe Anna, Smeeding James E, Lawrence Edwards N, Chronic gout: epidemiology, disease progression, treatment and disease burden, Current Medical Research and Opinion, 2010 Page No- 2813-2821.

[4] Perez-Ruiz Fernando, Dalbeth Nicola, Bardin Tomas, A Review of Uric Acid, Crystal Deposition Disease, and Gout, Advances in Therapy 2015 Page No- 31-41.

[5] Eggebeen, Aaron T, Gout: An Update, American Family physician 2007 Page No-801-808.

[6] Kumar, R., Saha, P., Kumar, Y., Sahana, S., Dubey, A., & Prakash, O. (2020). A review on diabetes mellitus: type1 & Type2. *World Journal of Pharmacy and Pharmaceutical Sciences*, *9*(10), 838-850.

[7] Saha, P., Kumar, A., Bhanja, J., Shaik, R., Kawale, A. L., & Kumar, R. (2022). A review of immune blockade safety and antitumor activity of dostarlimab therapy in endometrial cancer. *International Journal for Research in Applied Sciences and Biotechnology*, *9*(3), 201-209.

[8] Nyarko, R. O., Roopini, R., Raviteja, V., Awuchi, C. G., Kumar, R., Faller, E. M., ... & Saha, P. (2022). Novel Sars-CoV-2 Variants & Therapeutic Effects. *Journal for Research in Applied Sciences and Biotechnology*, *1*(2), 25-34.

[9] Awuchi, C. G., Saha, P., Amle, V. S., Nyarko, R. O., Kumar, R., Boateng, E. A., ... & Asum, C. (2023). A Study of various medicinal plants used in ulcer treatment: A review. *Journal for Research in Applied Sciences and Biotechnology*, *2*(1), 234-246.

[10] Sultana, A., Singh, M., Kumar, A., Kumar, R., Saha, P., Kumar, R. S., & Kumar, D. (2022). To identify drug- drug interaction in cardiac patients in tertiary care hospitals. *Journal for Research in Applied Sciences and Biotechnology*, *1*(3), 146-152.

[11] Kumar, S., Keshamma, E., Trivedi, U., Janjua, D., Shaw, P., Kumar, R., ... & Saha, P. (2022). A meta analysis of different herbs (leaves, roots, stems) used in treatment of cancer cells. *Journal for Research in Applied Sciences and Biotechnology*, *1*(3), 92-101.

[12] Kumar, R., Keshamma, E., Kumari, B., Kumar, A., Kumar, V., Janjua, D., & Billah, A. M. (2022). Burn injury management, pathophysiology and its future prospectives. *Journal for Research in Applied Sciences and Biotechnology*, *1*(4), 78-89.

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

[13] Kumar, A., Katiyar, A., Gautam, V., Singh, R., & Dubey, A. (2022). A comprehensive review on anti-cancer properties of Amaranthus viridis. *Journal for Research in Applied Sciences and Biotechnology*, *1*(3), 178-185.

[14] Kumar, R., Jangir, D. K., Verma, G., Shekhar, S., Hanpude, P., Kumar, S., ... & Kanti Maiti, T. (2017). S-nitrosylation of UCHL1 induces its structural instability and promotes α-synuclein aggregation. *Scientific reports*, *7*(1), 44558.

[15] Kumar, R., Register, K., Christopher-Hennings, J., Moroni, P., Gioia, G., Garcia-Fernandez, N., ... & Scaria, J. (2020). Population genomic analysis of Mycoplasma bovis elucidates geographical variations and genes associated with host-types. *Microorganisms*, *8*(10), 1561.

[16] Kumar, S., Yadav, S. P., Chandra, G., Sahu, D. S., Kumar, R., Maurya, P. S., ... & Ranjan, K. (2019). Effect of dietary supplementation of yeast (Saccharomyces cerevisiae) on performance and hemato-biochemical status of broilers.

[17] Hanna, D., Kumar, R., & Banerjee, R. (2023). A metabolic paradigm for hydrogen sulfide signaling via electron transport chain plasticity. *Antioxidants & Redox Signaling*, *38*(1-3), 57-67.

[18] Keshri, S., Kumar, R., Kumar, D., Singhal, T., Giri, S., Sharma, I., & Vatsha, P. (2022). Insights Of Artificial Intelligence In Brain Disorder With Evidence Of Opportunity And Future Challenges. *Journal of Pharmaceutical Negative Results*, 10853-10867.

[19] Kumar, A., Uniyal, Y., & Kumar, R. (2022). Recent Advancement of Colorectal Cancer and Their Herbal Essential Oil Treatment. *Journal for Research in Applied Sciences and Biotechnology*, *1*(5), 133-144.

[20] Chaudhary, H., Sagar, S., Kumar, R., Bisht, V., & Butola, K. (2022). Herbal Essential Oil use as Ulcer Protective Activity: A Systematic Review. *Journal for Research in Applied Sciences and Biotechnology*, *1*(5), 86-101.

[21] Kashyap, N., Kumar, R., Rana, V., Sood, P., & Chauhan, T. (2023). Role of Terpenoids Active Ingredients Targeting for Neuroprotective Agents. *Journal for Research in Applied Sciences and Biotechnology*, *2*(3), 22- 40.

[22] Raj, R., Kumar, A., Sood, P., Kumar, R., & Rana, V. (2023). Randomized Phase III Trial Comparing Epirubicin/Doxorubicin Plus Docetaxel and Epirubicin/Doxorubicin Plus Paclitaxel as First Line Treatment in Women with Advanced Breast Cancer. *Journal for Research in Applied Sciences and Biotechnology*, *2*(3), 55- 63.

[23] Kumar, R. (2023). Investigation of In-Vitro Method of Antiulcer Activity. *Journal for Research in Applied Sciences and Biotechnology*, *2*(1), 264-267.

[24] Borghi Claudio, The management of hyperuricemia: back to the pathophysiology of uric acid, Current Medical Research and Opinion 2017, Page No- 1-4.

[25] Kanwal Ashiq, Abida Latif, Sana Ashiq , Ahlam Sundus, A systematic review on the prevalence, pathophysiology, diagnosis, management and treatment of gout (2007-2018), GSC Biological and Pharmaceutical Sciences, 2018, Page No- 50- 55. Khan

[26] Akram Muhammad, Usmanghani Khan, Ahmed Iqbal, Azhar Iqbal, and Hamid Abdul, Comprehensive review on therapeutic strategies of gouty arthritis, Corresponding author 2014, Page No- 1575-1582.

[27] Robinson Philip C and Dalbeth Nicola, Advances in pharmacotherapy for the treatment of gout, Expert Opinion on Pharmacotherapy 2014 Page- 1-4.

[28] Roddy E and Doherty M, Epidemiology of Gout, Arthritis Research and Therapy 2010,12:223

[29] Aung Thanda, Myung Gihyun, and FitzGerald John D, Treatment approaches and adherence to uratelowering therapy for patients with gout, Patient Preference and Adherence 2017, Page No- 795-800.

[30] Kapoor Bhupinder, Kaur Gagandeep, Gupta Mukta, Gupta Reena, Indian Medicinal Plants In Treatment of Gout: A Review For Current Status and Future Prospective, Asian Journal of Pharmaceutical and Clinical Research 2017, Page No- 1-10.

[31] Ashiq Kanwal, Hussain Khalid, Islam Muhammad, Shehzadi Naureen, Ali Ejaz, Ashiq Sana, Medicinal plants of Pakistan and their xanthine oxidase inhibition activity to treat gout: a systematic review, Turkish Journal of Botany, 2021,Page No- 1-18.

[32] Abd El-Rahman Hanna S.M. and Abd- El Hak Nasra A.M., Xanthine Oxidase Inhibitory Activity and Antigout of Celery Leek Parsley and Molokhia, Advances in Biochemistry 2015, Page No- 1-11.

[33] Soliman Mohamed, Nassan Abdo Mohamed, Aldhahrani1 Adil, Althobaiti Fayez and Mohamed Abdou Wafaa, Molecular and Histopathological Study on the Ameliorative Impacts of Petroselinum Crispum and Apium Graveolens against Experimental Hyperuricemia, Nature research 2020, page no-1-11.

[34] Karim Abdul, Ali Bhatti Shabir M., Johnson Noman, Akhter Mahreen, Mona Sundus and

ISSN: 2583-4053

Volume-3 Issue-6 || December 2024 || PP. 33-40

https://doi.org/10.55544/jrasb.3.6.5

Safder Zartasha, Effect of Apium Graveolens (Celery) Seed Extract on Serum Uric Acid Level of Hyperuricemic Rats and its Comparison with Allopurinol, Journal of Shalamar Medical and Dental College, 2021 Page No- 1-7.

[35] Bibi Hafsa, Shahid Tahreem and Ain Quratul, Search for the Natural Remedies for the Treatment of Gout, Phytopharmacological Communications 2022 Page No- 193-204.

[36] Singh Akshay K, Srivastava Anjali, Kumar Vivek and Singh Karunakar, Phytochemicals, Medicinal and Food Applications of Shatavari (Asparagus racemosus): An Updated Review, The Natural Products Journal, 2018, Page No- 32-44.

[37] Bryan T and Emmerson, The Management of Gout ,Drug Therapy ,CORE 1996, Page No- 445-449.

[38] Eggebeen , Aaron T, Gout: An Update , American Family physician 2007 Page No-801-808.

[39] So Alexander , Developments in the Scientific and Clinical understanding of Gout , Arthritis Research and Therapy 2008, 10:221

[40] Martinon Fabio and Glimcher Laurie H, Gout: new insights into an old disease, The Journal of Clinical Investigation 2006 , Page No- 2073-2075.

[41] So Alexander and Busso Nathalie, Mechanisms of inflammation in Gout, Arthritis Research and Therapy 2010, 12:206

[42] Doherty Michael, Jansen Tim L, Nuki George, Pascual Eliseo, Perez-Ruiz Fernando, Punzi Leonardo, So Alexander k, Bardin Thomas, Gout: Why is this Curable disease So Seldom Cured? Annrheumdis 2012 Page No- 1-6.

[43] Cronstein Bruce N and Terkeltaub Robert, The inflammatory Process of gout and its treatment, Arthritis Research and Therapy 2006 Page No- 1-7.

[44] Major Tanya J, Dalbeth Nicola, Stahl Eli A and Merriman Tony R, An update on the genetics of hyperuricaemia and Gout, Nature Reviews Rheumatology 2018, Page No- 1-13.

[45] Monu Johnny U.V. ,Pope Thomas L, Gout: a clinical and radiology review, Radiologic Clinics of North America 2004 , Page No- 169-184.

[46] Saag Kenneth G and Choi Hyon, Epidemiology, risk factors, and Lifestyle modifications for gout , Arthritis Research and therapy 2006 Page No- 1-7.

[47] Malik A, Schumacher HR, Dinnella JE, Clayburne GM: Clinical diagnostic criteria for gout: comparison with the gold standard of synovial fl uid crystal analysis. J Clin Rheumatol 2009, 15:22- 24

[48] Roddy E: Hyperuricemia, gout, and lifestyle factors. J Rheumatol 2008, 35:1689-1691.

[49] Emmerson BT. The management of gout. N Engl J Med 1996;334 Page No- 445 – 451.

[50] Heber D. PDR for Herbal Medicines, 4th ed. Thomson Healthcare Inc.; 2007. 1026 p.

[51] Maiuolo, J., Oppedisano, F., Gratteri, S., Muscoli, C. & Mollace, V. Regulation of uric acid metabolism and excretion. Int. J. Cardiol. 213,Page No- 8–14.

[52] Dolati, K. et al. Inhibitory Effects of Apium graveolens on Xanthine Oxidase Activity and Serum Uric Acid Levels in Hyperuricemic Mice. Preventive Nutr. food Sci. 23,Page No- 127–133.

[53] Harris, Siegel LB, Alloway JA. Gout and hyperuricemia. Am Fam Physician 1999 Page No- 925 – 934.

[54] Terkeltaub R: Pathogenesis and treatment of crystal-induced inflammation. In Arthritis and Allied Conditions, 15th ed. Edited by Koopman WJ, Moreland LW. Philadelphia: Lippincott, Williams and Wilkins; 2004 Page No- 2357-2372.

[55] Terkeltaub RA: Clinical practice. Gout. N Engl J Med 2003, 349: 1647-1655.

[56] Choi HK, Mount DB, Reginato AM: Pathogenesis of gout. Ann Intern Med 2005, 143:499-516.

[57] Choi, H.K., Atkinson, K., Karlson, E.W., Willett, W. & Curhan, G. Purine- rich foods, dairy and protein intake, and the risk of gout in men. N Engl J Med 350, 1093-103 (2004).

[58] Merriman, T.R. An update on the genetic architecture of hyperuricemia and gout. Arthritis Res Ther 17, 98 (2015).

[59] Dalbeth N, Haskard DO. Mechanisms of inflammation in gout. Rheumatology (Oxford) 2005;44:Page No- 1090–6.

[60] Simkin PA. Gout and hyperuricemia. Curr Opin Rheumatol 1997;9(3): Page N - 268 – 273

[61] Asplin JR. Uric acid stones. Semin Nephrol 1996; 16(5): Page No- 412-424