International Journal of Research and Review
Volume 11; Issue: 6; June 2024
Website: www.ijrrjournal.com
Review Article *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

Gouty Arthritis and Its Appraisal in *Greco-Arab* (Unani) Medicine

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DOI: https://doi.org/10.52403/ijrr.20240688

ABSTRACT

A typical excruciating type of inflammatory arthritis, Gout, often affects big toe joint and sometimes other joints and viscera as well. In Greco-Arab (Unani) medicine gout is known as *Niqris*. There is ample literature regarding gouty arthritis available in the treatise of Greco-Arab medicine, an age old existing medicinal system. The aim of this study is to explore the medicinal treasure for gout in Greco-Arab medicine, correlate it with the modern science to amplify the knowledge and widen the field of research avenues. More efficient, cost-effective management of gouty arthritis with least side effects could be achieved with these medicines.

Key words: Arthritis, Gout, *Niqris*, Unani

1. INTRODUCTION

A typical excruciating type of inflammatory arthritis, Gout, often affects big toe joint and sometimes other toe joints, the ankle, and the knee joint as well. There are remissions and flares, or periods without symptoms. It is a condition when an excess of uric acid in the body leads to the formation of crystals of monosodium urate (MSU) in joint fluid, bones, cartilage, tendons, bursa, and other tissues.[1] In Greco-Arab (Unani) medicine gout is known as *Niqris*. According to Ibn-e-Hubal, a Greco-Arab physician, the word Niqris originated from the word 'Anquroon' which means big toe, because this disease usually starts with the involvement of metatarsophalangeal joint and hence the disease has been named after this joint.[2]

2. Historical background

Early in the 1200s, a Dominican monk named Randolphus of Bocking used the word "Gout" for the first time. The word "Gout" came from Latin *"gutta",* which means "drop".[3] Back then, it was considered that when one of the four "*humors*," or body fluids, flowed or "dropped" into a joint in excess, it would result in pain and inflammation[4]. The name continues to be used today in many other languages, including English ("gout"), French (*"goutte*"), Spanish (*"gota*"), Italian ("*gotta*"), and German (*"gicht*") [5] One of the first illnesses known was Gout, which was first recognized by the Egyptians in 2640 BC.[3] Gout was originally mentioned in writing by the Egyptians as *podagra* (foot pain), or gouty arthritis, usually affecting the big toe and now known as uric acid arthropathy.[5,6] The first mention of this topic may be found in the Ebers Papyrus, which was written around 1500 B.C., where a medication that possibly corresponds to colchicine was mentioned. The Latin terms podagra (seizure in the foot) and chiragra

(seizure in the hand) were utilized by the Roman physician Celsus (25 B.C. - A.D. 50) to describe a condition resembling gout.[7,8,9]

Hippocrates described gout in the fifth century BC. as "the unwalkable disease." He added that elderly people with gout, have tophi in their joints are difficult to treat. He was first to mention gout as a genetic sickness and provided the first recognized clinical definition of gout.[2,10,11] Depending on whether the big toe, hand, knee, or shoulder gets affected, Hippocrates referred Gout as *podagra cheiragra, gonagra, or omagra* respectively.[6] He also revealed that gout does not develop in eunuchs, women till menopause and in men till they have had a sexual intercourse. [2,10,12]

In the fourth century B.C., Plato also discussed Gout, although he added nothing new to Hippocrates' theory. For a long time, Gout was viewed as largely a male illness. Roman senator Seneca emphasized the importance of heredity in gout in the first century A.D. Gout, according to Diocles of Carystus (4th century B.C.), is an inflammatory condition that develops when bad humors build up in the joints of the foot. Pedanius Dioscorides," the father of pharmacognosy" (Ist century A.D) in his work "De universa medicina" (*kitab-ul-Hashaish* in Arabic) address the application of colchicum in gout as its treatment. [4,13]

Roofas al-Afsi, a notable physician in the Roman era, in his renowned treatise *"Kitab-fil-auja-ul-mafāsil"* discussed the causes, clinical characteristics, and therapy of gout separately. Aretaeus (81–138 A.D.) reported polyarticular gout.[4,12,14] Around 200 A.D., Galen, described gouty tophi and asserts that coitus is a key contributor to gout, explaining why eunuch and young men are not affected by it.[12,15] Phythagorus, a great scholar of the Byzantine era (4th –5th century A.D.), wrote a booklet about gout called "*Risala-fi-Auja-Niqris."* He emphasized that the primary cause of gout is injury. Gout was treated with an infusion of the whole plant (colchicum autumnale) by Paul of Aegina in the seventh century A.D.[4,12] Rhazes (850-923 A.D.) a well-known Arab physician, in his books "Liber Continens" *(Al-Hāwi), Al-Mansoori,*and "*Al-Fakhir* discussed the clinical characteristics and management of Gout.[16,17]

In the well-renowned book "Canon of Medicine," Avicenna (980–1037), extensively discussed the genesis, clinical characteristics, and management of gout.[15,18,19]

Ali-Ibn-Abbās-Al Majoosi (930-994) described that gout is the type of discomfort that affects joints of one or both legs, the wrist or sometimes elbow, and mostly affects the joints of great toe. The author of the well-known book "*Kitab ul Mait," Abu Sahel Maseehi*, detailed the etiology of gout in the 10th century A.D.[11,20]

Gout is described by *Ismail Jurjāni* (11th century AD) in "*Zakheera Khwarzam Shāhi"* as morbid humors that accumulates in the small joints and results in pain and inflammation. It mainly affects the larger toe.[21,22] In the year 1290, the term "Gout" was first written in English.[23]

In about 1679, Antoni van Leeuwenhoek provided the description of the microscopic structure of tophi. This discovery is in line with current uric acid crystal microscopy and the use of the presence of uric acid crystals in joint aspirates as a diagnostic indicator for Gout.[24,25] Thomas Sydenham provided a thorough account of the acute disease's symptoms in 1683 based on his own experience as a doctor and a gout patient.[26,27]

William Stukeley reported the crystals from a tophaceous joint in 1734, and Scheele (a Swedish chemist) found uric acid 42 years later. Prof. Baron von Stoerk made a new discovery of colchicines in Vienna in 1763. Wollaston demonstrated in 1797 that tophi comprised of uric acid deposits. Nearly a century later, Mc Carty and Hollander employed polarized microscopy to

demonstrate that monosodium urate is present in the joint fluid of gout patients.[8,26] Sir Alfred Garrod identified the link between hyperuricemia and gout in 1848, and he published his findings in The Nature and Treatment of Gout and Rheumatic Gout in 1859. It was the first clinical chemical test ever conducted. Alfred Garrod described his renowned "thread test," a semi-quantitative method for the determination of uric acid in the blood or urine. It was not until 1859 that Sir Albert Baring Garrod hypothesized that" Urate deposit is the cause and not an effect of Gout". For the treatment of gout, he recommended a diet low in foods high in purine.[13,28] Freudweiler's (1899) experiment, which demonstrated that one could simulate an outbreak of acute gouty arthritis by injecting sodium urate crystal into joints, reinforced this.[27,28]

As a gout patient, Haig claimed to have performed multiple tests on himself between 1894 and 1897 to demonstrate that reducing the consumption of purine-rich meals helped lessen hyperuricemia.[29,30]

Huber provided the first radiological description of gout in 1896, while Emil Fischer provided the disease's pathogenic mechanism in 1898. Folin and Denis established the serum urate measurement in 1913. Sir Archibald Garrod proposed that gout be classified as an illness with an in-born metabolic defect in 1931.[31,32] Aspirin was used to treat gout by the 1940s. In 1950, Talbot, Gutman, and Yu discovered probencid's uricosuric action, while allopurinol was first used to treat gout in 1963. For creating allopurinol, azathioprine, and other medicines, George Hitchings and Gertrude Elion were given the Noble Prize in 1988. The first-time arthritis linked with articular crystal deposition was recognized when Mc Carty and Hollander discovered monosodium urate (MSU) crystal in the synovial fluid of a patient with severe gout in 1961.[33,34] In 1967, Seegmiller, Rosenbloom, and Kelly identified the first enzymatic flaw (HGPRT deficiency) causing a subtype of adult primary gout. Sperling and his coworkers found the second enzymatic flaw five years later, in 1972. (PRPP synthetase overactivity in urate overproduction).[35] Klinenberg proposed in 1977 that gout patients may be roughly divided into three groups: overproduction, underexcretion, or a combination of the two abnormalities.[26,28] With the development of prednisolone (1995), indomethacin (1999), and ACTH (adrenocorticotropic hormone), the middle of the 20th century was perhaps the halcyon days of gout therapy.[34]

However, gout is still the most prevalent inflammatory arthritis in males over 40 in the twenty-first century, and cases are rising in all racial groups.

3. Epidemiology

Gout is most prevalent inflammatory arthritis. Its global incidence ranges from 0.1% to 3.0%.[36] Gout is becoming more common in the world on a steady basis due to poor eating habits such fast food, inactivity, rising obesity rates, and metabolic syndrome.[37] The incidence of gout rises to 11–13% and the incidence rises correspondingly with each decade of life. In adults above the age of 80, the incidence rises to 0.4% [38] According to the NHANES survey, there are roughly 9.2 million gout sufferers in the United States (U.S.), or 3.9% of the population. According to Rochester Epidemiology[39], the prevalence of primary gout increased over the previous 20 years. According to certain studies, urate is inherited between 45% and 73% of the time.[40, 41]

The frequency of gout found in these researches varies noticeably. This is most likely explained by a variety of factors, including different sampling and case-finding and definition techniques, different estimation times for prevalence (confined-period prevalence vs. lifetime prevalence),

demographics, and variations in genetic, lifestyle, and co-morbid risk factor profiles among populations from various geographic regions. Although this methodological and clinical variability still has an influence, comparing estimates from the same countries and datasets can mitigate this effect and still show that gout has been more prevalent over the past few decade.[42,43]

Globally, reports of the prevalence and incidence of Gout range from 0.1% to around 10% and from 0.3 to 6 cases per 1,000 person-years, respectively.[36] According to a recent study, gouty arthritis affects 4-6% of males and around 2% of women in western nations including Europe, the United States, Canada, Australia, and England.

According to a study done in the northern hemisphere, spring is the time of year when acute gout flares occur most frequently. Arber et al. found a higher incidence of gout in the spring and summer, whereas Mcleod reported in 1972 that the majority of Australian gout patients experienced symptoms in the autumn. The spring season is when Tophaceous gout is believed to occur most frequently.[44,45]

4. Etiopathology

According to modern science gout is a form of inflammatory arthritis that is brought on by uric acid crystals in the joints and is frequently linked to hyperuricemia. The pathogenesis of gout has been thoroughly characterized in contemporary medicine. An increase in urate synthesis and its decreased excretion results in supersaturation and accumulation of crystals in joints hence the inflammation (figure 1)[46,47]

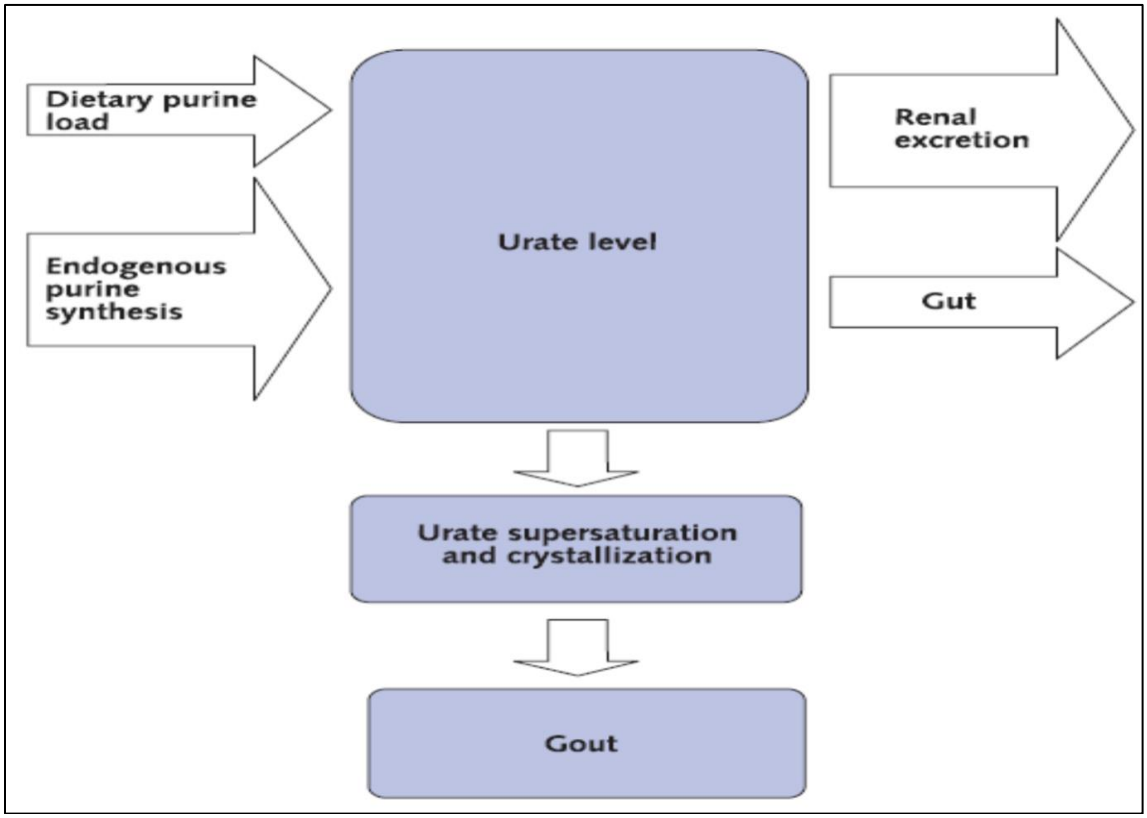

Figure-1: Etiopathology of Gout

5. Risk factors

Obesity, hypertension, medication (loop and thiazide diuretics), diet heavy in purines, alcohol, etc. are all modifiable risk factors, whereas age and gender are regarded as non modifiable risk factors for Gout.[42] It primarily

occurs after 40 years and is 10 times more common in men than in women.[48,49]

6. Classification

Gout has been classified into various types depending upon causes, severity and other complications; [50]

6.1 Dorland's Classification:

a) Latent gout
b) Oxalic gout
c) Polyarticular gout
d) Rheumatic gout
e) Saturning gout

6.2 Primary and Secondary gout

Depending on whether a known cause of hyperuricemia is present or absent, gout can be categorised as primary or secondary. Therefore, primary gout is neither a consequence of an inherited condition nor the outcome of a congenital flaw. Obesity, alcohol use, hypertension, and hypertriglyceridemia are other disorders that frequently accompany primary gout and should be carefully evaluated. The use of some medicines can cause secondary gout, and it can also develop as a result of other conditions such as lead poisoning, renal failure, and, more specifically, the uncommon familial juvenile hyperuricaemic nephropathy and the autosomal dominant medullary cystic kidney.[51,52]

7. Clinical features

Studies in recent past have recognized four different clinical stages in the development of gout namely Asymptomatic Hyperuricemia, Acute gouty arthritis, Intercritical (interval) and Chronic tophaceous gout. [50,53]

7.1 Asymptomatic Gout

High blood urate levels are indicative of a disease, although gouty symptoms, tophi, or uric acid nephrolithiasis have not yet materialised. Most patients are asymptomatic for the majority of their lives.[31,51]

7.2 Acute gout

The symptoms typically associated with gout — severe pain, tenderness and swelling occur in the acute phase. Generally, on initial presentation, only one joint is affected by acute gout (monoarticular arthritis), and in 50% of cases the first metatarsophalangeal joint is the one involved. Other joints commonly affected include the foot, ankle, knee, wrist, finger and elbow. These peripheral joints are thought to be more susceptible due to their cooler physiological temperature, which facilitates urate crystallization.[54] An episode of acute gout often begins in the early hours of the morning. Often, the first symptom patients will describe is an itching sensation, thought to be caused by prodromal mast cell degranulation and release of histamine.[55]

7.3 Inter-critical or interval gout

Once the first episode of acute gout resolves, usually in seven to 14 days, a symptom free period will follow — this is termed "intercritical gout". The duration of this asymptomatic phase can vary widely from several days to years. Although some patients will never have another attack, for most the second attack occurs within a year. [56,57]

7.4 Chronic tophaceous gout:

The most disabling stage of the condition is chronic tophaceous gout, which usually develops over an extended period. Chronic tophaceous gout has become rare since the introduction of effective long-term prophylactic therapy. This stage of gout is characterised by clinically apparent tophi (subcortical cysts) that form within joints previously affected by acute gout. Chronic gout tends to be more of a problem for patients in whom prophylactic therapy is contraindicated or poorly tolerated (e.g., those

with allopurinol hypersensitivity or chronic kidney disease). Chronic tophaceous gout causes permanent damage to the affected joints. Kidney damage is also common in this stage.[58,59]

8. Complications

Like rheumatism, gout is not lethal in itself, but it can quickly cause death by altering the structure of the kidneys, liver, heart, and other internal organs. The heart and blood vessels are the most common sites of this illness, which is medically known as fibroid degeneration. The arteries become stiff and rigid, making them susceptible to unexpected rupture from minor causes. Apoplexy, a common mishap among gouty people, is referred to as when this rupture happens inside the brain and causes haemorrhage. People with gout frequently have pulmonary conditions such chronic bronchitis and asthma. [23,60]

9. Diagnosis

gout patients may appear with mono or polyarticular acute, subacute, or even chronic arthritis rather as the more typical inflammatory monoarthritis or inflammatory oligoarthritis. [61,62]

9.1 Serum Uric Acid levels

Nearly all cases of gout can be diagnosed by a high blood uric acid level and a typical history. Up to 10% of gout patients have normal blood uric acid levels when they are having an attack.[61,63]

9.2 Radiographic Features

Although radiographs rarely help in the diagnosis of acute gout, they can prove useful in verifying alternative diagnoses such chondrocalcinosis. [64]

9.3 Ultrasound

Sonography is more capable of detecting subtle changes. Sonography can identify tophaceous debris, common erosions, and MSU crystal deposition on cartilaginous surfaces.[61,65]

9.4 Synovial Fluid Aspiration

Leukocyte numbers in synovial fluid are increased from 2000 to 60,000/L. Effusion appears hazy because of the elevated leukocyte count. MSU crystals are frequently found in the first metatarsophalangeal joint and in acutely gout-affected knees.[20]

10. Management

Avoid sedentary and luxury life style, alcohol intake, excessive eating and physical inactivity.

10.1 Pharmacotherapy

Different types of medicines used to treat gout include;

I. Uricostatic drugs (Decrease production of uric acid): Allopurinol

II. Uricosuric drugs (Increases excretion of uric acid via kidney): Probenecid, Sulphinpyrazone

III. Inhibit neutrophil migration into joint: Colchicine

IV. Inhibit inflammation and pain: NSAIDs

V. Uricolytic (Increasing uric acid oxidation): Urate oxidase

11. Appraisal of Gout (*Niqris*) in Greco-Arab system of medicine

According to Greco-Arab (Unani) physician, Avicenna, the matter causing gout (*Mādda*-e-*niqris)* can be blood or can be combination of blood and phlegm or blood and yellow bile or blood and black bile or purely phlegm.[66,67,68] *Rāzi* stated that raw phlegm is the cause of gout.[10]

11.1. *Asbab wa Darja bandi* (Etiology and Classification)

In the thirteenth century, Hippocrates and Randolphus of Bocking believed that gout was caused by an abnormal accumulation of one of the four *humors*, black bile, yellow bile, blood, and phlegm, in the joints.

Gout can be divided into four basic forms, according to Hakeem Ghulam Jeelani;
Acute (*Niqris shadeed*), chronic (*Niqris muzmin*), visceral (*Niqris dakhli*), and intermittent (*Niqris beqaida).*[69]

11.2. *Tahaffuz* (Prevention)

Unāni physicians have recommended the following preventive measures of gout;
Avicenna has recommended that patients suffering from gout should strictly avoid meat.[70]
Nooh- bin-Mansoor Qamri stated that Patients suffering from gout should avoid intake of meat, alcohol and should increase the intake of water.[70]
Zakariya Rhāzi recommends that patients of gout should avoid eating beef and other kinds of meat. Dairy products and certain types of dried fruits *viz.* walnuts, dried dates, unripe dates, honey, pine seeds, Syrian carob beans, should be avoided.[71] Excessive sexual intercourse is also prohibited.[71]

11.3. *Usool-e-ilāj* (Principle of treatment)

Correction of *So'e Mizāj* (unbalanced temperament) by appropriate Measures.[61,67]
Evacuation of the morbid matter (causative humor) by different means like the use of *Munzijat* (concoctants), *Moarriqāt* (diaphoretics), *Mushilaat* (purgatives), *and Muqiyāt* (emetics) or *Mudirāt* (Diuretics).[17,20,66]
Use of *Mohallil-e-aurām wa musakkin alam* (anti-inflammatory and analgesics) drugs both in systemic & local forms.[20,66]

11.4. *Ilaj* (Treament)

11.4.1. *Ilaj bil tadbeer* (regimental Treatment)

Ali Ibn-e-Zain stated that walking bare-footed during summer seasons is beneficial to the gouty patients.[70] According to Hippocrates pouring cold water on feet from certain height is favorable to the gouty patients.[20,66] Similarly Dascaridoos recommends pouring sulfur water is benificial.[17]

11.4.2. *Ilaj Bil Ghiza* (Dieto Therapy)

Consumption of easy to digest diet (*zud hazm* ghiza) i.e. *chapati*, milk, rice, barley water, spinach (*spinacia oleracea* l.), fenugreek (*trigonella foenum-graecum* l.), pear (*pyrus communis* l.) and apple juice is recommended.[17]

11.4.3. *Ilaj bil advia* (Pharmacotherapy)

a) Single drugs

Some single drugs used in gout since ancient times are; *Banafsha (Viola odorata) , Barg-i-Karnab (Lactuca sativa) , Halela Zard (Terminalia chebula) , Khitmi (Althea officinalis), Saqmonia (Convolvulus scammonia), Shahm Hanzal (Cytrullus colysinthis),Sibr (Aloe vera), Shitraj Hindi (Plumbago zylanica) , and Suranjan (Cholchicum luteum). Colchicum luteum* hydroalcoholic extract (CLHE) produced a significant and dose dependent inhibition of joint swelling. Serum TNF-α level and pro-inflammatory mediators (TNF-R1, IL-6 and IL-1β) were also reduced significantly in a dose dependent manner. It shows that the anti arthritic activity of Colchicum luteum is due to its modulatory effect on the expression of pro inflammatory cytokine in the synovial fluid.[20]

b) Compound drugs

Habb-i-leemun, Habb-i-Suranjan, Jawarish Zar"uni Ambarin Ba Nuskha Kalan, Majun Suranjan, Bonigra capsule, Rogha-i-Shifa, Roghan Surkh, Roghan Mufasil etc are best known Unani compound formulations used for gouty arthritis.[17,20]

12. CONCLUSION

Unani medicinal system has broadly discussed gout since ancient times and mentions potent medicinal herbs, regimental therapies besides robust preventive measures

and ditary advisory for gout. Thus it provides a vast ground for research and cheap, effective and safer alternative for gout management which needs to be evaluated scientifically.

Declaration by Authors

Ethical Approval: Not Applicable
Acknowledgement: None
Source of Funding: None
Conflict of Interest: The authors declare no conflict of interest.

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How to cite this article: Asma Afzal, Shameem Ahmad Rather, Shabir Ahmad Bhat. Gouty arthritis and its appraisal in *Greco-Arab* (Unani) medicine. *International Journal of Research and Review*. 2024; 11(6): 818-827. DOI: *https://doi.org/10.52403/ijrr.20240688*

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