

INTERNATIONAL JOURNAL OF UNANI AND INTEGRATIVE MEDICINE



E-ISSN: 2616-4558
P-ISSN: 2616-454X
www.unanijournal.com
IJUIM 2025; 9(2): 277-283
Impact Factor (RJIF): 6.59
Peer Reviewed Journal
Received: 14-06-2025
Accepted: 19-07-2025

Arshiya Begum
PG Scholar, Department of
Ilmul Advia (Pharmacology),
Government Nizamia Tibbi
College, Charminar,
Hyderabad, Telangana, India

Zaibunissa Begum
H.O.D & Professor,
Department of Ilmul Advia
(Pharmacology), Government
Nizamia Tibbi College,
Charminar, Hyderabad,
Telangana, India

Ather Moin Rasheeda
Assistant Professor,
Department of Ilmul Advia
(Pharmacology), Government
Nizamia Tibbi College,
Charminar, Hyderabad,
Telangana, India

Corresponding Author:
Arshiya Begum
PG Scholar, Department of
Ilmul Advia (Pharmacology),
Government Nizamia Tibbi
College, Charminar,
Hyderabad, Telangana, India

Niqris (Gout): A comprehensive review of concepts and management in modern and Unani medicine

Arshiya Begum, Zaibunissa Begum and Ather Moin Rasheeda

DOI: <https://www.doi.org/10.33545/2616454X.2025.v9.i2d.370>

Abstract

Gout, also called *Niqris* in Unani medicine, is a type of inflammatory arthritis that most often targets the great toe (metatarsophalangeal joint). *Niqris* is a term based on the Unani word *Inqurus* which as *Ibn-e-Habal* refers the joint of the big toe where this condition is most often found to emphasize the common location of the original attack. In modern medicine, gout is the result of increased purine metabolism or decreased renal excretion, but in Unani medicine the cause of *Niqris* (gout) is the imbalance of humors (*Akhlaat*) caused by either *Dam* (Blood), *Balgham* (Phlegm), *Safra* (Yellow bile) or *Sawda* (Black bile), or a combination of humors. The aims of this review were to compare concepts, pathogenesis, clinical features and treatment of gout in the modern and Unani system of medicine and point out areas where the two systems converged and diverged. A thorough review of the classical Unani texts, modern medical and recent scientific literature was carried out. Data synthesis was carried out to compare the conceptual framework, diagnostic profiles and treatment modalities articulated in both systems.

The focus of modern medicine is on pharmacological agents, such as NSAIDs, colchicine, corticosteroids, urate-lowering treatments, and supplemented by lifestyle changes. Unani medicine, in contrast, is holistic involving *Ilaj bi'l Ghidha* (dietotherapy), *Ilaj bi'l Dawa* (pharmacotherapy with herbal medicine) and *Ilaj bi'l Tadbeer* (regimenal therapy) in achieving detoxification and humoral equilibrium. A comparative study shows that the understanding and management of gout are complementary through two different and overlapping perspectives of Unani and modern medicine (*Niqris*). Evidence based pharmacological treatment based on integrative approach, including Unani principles of lifestyle control and detoxification, can yield better long-term results in the management of gout. The classical Unani therapies should be validated by further research using modern clinical trials.

Keywords: Gout, Niqris, Hyperuricemia, Unani medicine, integrative management, uric acid

Introduction

• Historical Background

One of the oldest known diseases in human history, gout appeared in the writings of Hippocrates as early as the 5th century BCE. It began to be referred to as the disease of kings or the disease of rich men, and it was linked to indulgence in meat and alcohol. In Unani medicine, the illness is referred as *Niqris*, and it is explained by such great scholars like *Buqrat* (Hippocrates), *Jalinus* (Galen), *Ibn Sina* (Avicenna), and *Zakariyya Razi* (Rhazes). These traditional doctors described its etiology, clinical features, preventive and treatment interventions in humoral terms. Modern and traditional views of gout, therefore, have long considered gout a prominent lifestyle and metabolic disorder.

• Epidemiology and Clinical Significance

Gout occurs more in developed nations than developing nations but its prevalence is on the increase in all nations. Hyperuricemia is the key cause of gout. Despite this, a number of other factors cause its increasing prevalence among which are: genetic predisposition, some medications and dietary habits ^[1]. Gout itself has a prevalence of about 4% of the U.S. population approximately 9.2 million people; as compared to hyperuricemia, the condition or state of high levels of uric acid in the blood, which has a prevalence of about 20% (or 46 million people). Both conditions have been increasing in the United States and other western countries over the last few decades. This is because of sharp rise in obesity and metabolic syndrome which are a well-known risk factors.

This is also more prevalent in men than women. Indeed, gout is very uncommon in premenopausal females, partly because estrogen has uricosuric (Reducing uric acid) properties. Mono sodium URATE crystals may develop and deposited in joints and the surrounding tissues when blood levels of uric acid remain consistently at or above 6.8 mg/dL, causing gout attacks. Both the duration and the degree to which one has an increased level of uric acid are closely associated with the risk of developing gout [12].

• Modern Concept of Gout

Gout is a disorder of uric acid metabolism, characterised by deposition of urate crystals in the tissues, especially in the joints, and by an increase in the concentration of uric acid in tissue fluids, including the blood plasma. The disease is characterised by recurrent attacks of acute arthritis, often in the big toe. Precipitation of excess urates occurs in the synovium of the joints and in the surrounding connective tissue [13].

• Unani Concept of Nigris

The *Nigris* is a systemic disorder caused by the imbalance of humors (*Akhlaat*) in the Unani system of medicine. Excess humors accumulation that concentrates and localize in peripheral joints is the main cause of pain, swelling and dysfunction. Unani doctors pointed out the disease was an expression of imbalance in the humors and temperament and not just limited to joint pathology. Management is thus holistic and it includes:

- ***Ilaj bi'l Ghidha* (diet therapy):** Here the focus is laid on diet and the use of food and nutrition to balance the humors of the body and facilitate healing.
- ***Ilaj bi'l Dawa* (Pharmacotherapy):** The treatment of illnesses by using herbs and natural medicines to restore balance in the body's humors
- ***Ilaj bi'l Tadbeer* (regimenal therapy):** This is a type of physical treatment that includes exercises, bathing, massage, etc., to repair the balance and enhance general health.

• Reason to Compare

Even though currently there are specific pharmacological treatment approaches of modern medicine, there are still safety issues and the reoccurrence of the disease. Unani medicine offers a preventive and holistic approach to medicine based on humoral balance, detoxification and lifestyle control. Bridging these two perspectives can enrich clinical understanding and create new possibilities in integrative management. This review thus aims to compare the concepts, pathogenesis, clinical presentation, and treatment of *Nigris* (Gout) in Unani and modern medicine, with respect to their complementary and integrative possibilities.

Historical Perspectives

Gout in ancient medicine

Gout was already known to the ancient Egyptians under this modern meaning in 2640 B.C [14]. The term gout was first applied in the modern meaning by Dominican monk Randolphus of Bocking (1197-1258) who utilized it to describe painful swelling commonly found in the big toe. Gout is derived from Latin (*gutta*) which means drop as excess of any of the four humors moves into a joint, distending it, and causing pain [15]. In unani medicine, gout is

referred to as “*Nigris*” [6]. The term *Nigris* is a derivation of the word *Inqurus*, which (as *Ibn-e-Habal* understands it) refers to the joint of the big toe, where the condition usually starts [7, 8]. One of the earliest to differentiate between the various forms of joint diseases (such as gout and rheumatic arthritis) was Hippocrates, who wrote in the 5th century BCE. He described a word *podagra*, combining the Greek *pous* (foot) and *agra* (trap) to refer to severe foot-based pain that makes the foot feel like it was caught in a trap. Similar conditions of other joints were given greek names as well: *chiagra* (hands) and *gonagra* (knees). Hippocrates believed that *podagra* was the worst joint disorder, it was usually chronic and centered in the great toe. He referred to it as unwalkable disease since it causes walking difficulty [5].

He outlined five aphorisms related to gout:-

- Eunuchs do not suffer from gout nor become bald.
- Women rarely develop gout unless their menstruation ceases.
- Young men typically don't get gout before engaging in sexual activity.
- Gout-related inflammation usually resolves within 40 days.
- Gout tends to become more active in spring and autumn [9].
- Traditionally also referred to as the disease of kings, gout was seen as a sign of wealth and luxury especially over-indulgence in excessive food and alcohol, which were a luxury of the rich [18].

• Concept of Nigris in unani literature

Ibn Sina (Avicenna) described *nigris* as a form of arthritis that begins in the joints of the foot, particularly the big toe, but may also begin in the heel or in the lower surface of the foot. In other circumstances, the condition starts in a single side and slowly extends to all parts of the foot and in some instances up to the thigh. The inflammation extends beyond the joints, it may also affect adjacent tissues which include nerves, ligaments and tendons and is thus a complex and painful condition [10].

Al-Razi, reported that pain caused by *nigris* tends to start in one joint and then spreads. It normally begins in the toe but may also begin at the heel or sole. Sometimes both feet gets affected. Once the disease progress, it can spread to the calf muscles, knees, bladder and rectum. In extreme cases, swelling may occur in the calf and thigh, sometimes elongated testis may be observed in some patients [11].

Ibn Hubal Baghdadi, noticed similar observations who wrote that *nigris* tends to start in the first metatarsophalangeal joint (the joint of the big toe). The pain may, however, also begin around the ankle or just below the heel, but the big toe is most commonly affected [12].

Ali Ibn Abbas Al-Majoosi also explained *nigris* as a painful disease which mostly affects the joints especially the knee or the joints of the toes especially the big toe [13].

Jurjani elaborated that the condition was caused by the accumulation of excess morbid humors or *Mavad-e-Fazooni*, in the small joints of the body specifically in big toe, ankle, and smaller joints of the foot, which causes pain and inflammation [14].

• Evolution of understanding in modern medicine

Thomas Sydenham an English physician and often called as

English Hippocrates who himself suffered by gout chronicled the cyclical nature of gout attacks and differentiated it from other arthritic conditions ^[5, 16] Antoni van Leeuwenhoek (1632-1723), was the first to describe the crystals found in gouty tophi. Although the exact chemical nature of these crystals was not known Decades later, in 1776, Swedish chemist Carl Wilhelm Scheele isolated a new compound from urinary stones. In 1797, English chemist William Hyde Wollaston another gout sufferer found the same substance taken from a tophus in his own ear. Initially, this compound was referred to as "urolytic acid" ^[15, 16]. Nevertheless, in 1798 it was discovered by the French chemist Antoine de Fourcroy as a naturally occurring substance in urine and was named uric acid. In 1848 Alfred Baring Garrod observed the presence of high levels of uric acid in gout patients which laid the foundation of metabolic basis. He also proposed increased uric acid could result from either over production or reduced excretion which was later confirmed by B. Wyngarden and his colleagues. He also demonstrated that the deposition of uric acid crystals in or around joints can trigger the gout attacks. This was in turn supported by Max Freudweiler in 1899 ^[16].

Etiopathogenesis

• Modern Perspective

Several causes and risk factors exist for gout

- **Increased Diet:** Red meat, shellfish, high fructose sugar sweetened drinks.
- **Diminished renal excretion:** Increased renal tubular reabsorption, renal failure, lead toxicity, lactic acidosis, alcohol.
- **Drugs:** Thiazide and loop diuretics, Low-dose aspirin, Cyclosporine, Pyrazinamide, Cancer chemotherapy, ACE inhibitors
- **Increased production:** Lymphoproliferative and Myeloproliferative disease, Psoriasis, High fructose intake, Glycogen storage disease
- **Inherited disorders:** Lesch-Nyhan syndrome (HPRT mutations), Phosphoribosyl pyrophosphate synthetase 1 mutation

Research has shown that non-modified risk factors such as increased age, sex and modified risk factors such as diet, medications, obesity and several health conditions such as metabolic syndrome, heart disease, diabetes, high blood pressure, kidney disease, hyperlipidemia and menopause can increase a person's risk of developing gout ^[17-21].

Pathophysiology

The food we eat contributes about one-third of the body uric acid, the remaining two-thirds are made by the body in the process of breaking down purines. The amount of uric acid in the body depends on production to excretion ratio (about two-thirds of it is excreted through the kidney), and the remainder is excreted through the gastrointestinal tract. The synthesis and degradation of purines is a highly interconnected enzymatic process. Of these enzymes, xanthine oxidase is of hugely significant importance. It breaks down hypoxanthine to xanthine, and finally to uric acid ^[17].

Pathogenesis

Hyperuricaemia is closely associated with pathogenesis of gout. Urate crystals in the joints stimulate an inflammatory

response when they build up in the joints, causing the release of cytokines that draw immune cells, such as leukocytes, into the area. The urate crystals are ingested by macrophages and neutrophils. This will cause an inflammasome (sensor located inside the cell) to become activated, which triggers an enzyme called caspase-1. Caspase-1 is essential in production of interleukin-1 β (IL-1 β). IL-1 β further increases the inflammation by attracting more immune cells into the joint. All these immune cells produce numerous inflammatory mediators like increased cytokines, free radicals, proteases, and arachidonic acid metabolites that cause tissue damage. The crystals may also rupture phagolysosomes, releasing the destructive contents of these phagolysosomes thus aggravating the inflammation. The complement system is also activated, especially via the alternative pathway, bringing more leukocytes to the location. This strong immune response leads to an acute gout attack, or arthritis, which eventually heals on its own in a few days to weeks. But in case gout attacks are frequent or persistent, chronic inflammation can occur. In the long term, this might result in the development of tophi hard lumps made of urate crystals and inflammatory tissue in both the joint and tissues around it. These tophi may cause serious harm to cartilage in the joints causing pain, stiffness, and joint dysfunction ^[22].

• Unani Perspective

Causes and risk factors

Imbalance of temperament, Imbalance of humour such as *Khilte balghami*, Sometimes *khilte safravi or damvi*, Deposition of chyme (Kaimoos) over the joints, Purine rich food, Indigestion, Excessive coitus, Alcoholic, Obesity, Cold temperature, Less exercise, Flatulence and Luxurious lifestyles ^[7, 8, 10-14, 19, 20, 26, 27].

Pathophysiology

In Unani medicine, many scholars believe that *Niqris* (gout) usually results from an imbalance in the body's humors (*Akhlat*) known as *Sue Mizaj Maddi*. Although a simpler form called *Sue Mizaj Sada* which refers to a general disturbance in temperament can also lead to gout, it is relatively uncommon and often easier to manage by restoring the body's natural balance. On the other side, *Sue Mizaj Maddi* is more complex and demands a thorough evaluation to determine which specific humor is out of balance before appropriate treatment can be planned ^[10, 12, 23].

Niqris (gout) development is explained through the imbalance of the body's humors. Food is processed through a series of natural faculties: *Quwat-e-Jazeaba* (the power of absorption), *Quwat-e-Maseka* (the power of retention), *Quwat-e-Hadima* (the power of transformation), and *Quwat-e-Dafiyah* (the power of elimination). If these processes particularly *Quwat-e-Hadima*, responsible for proper digestion and transformation do not function effectively, the food is not fully assimilated. Instead, it turns into harmful substances known as *fuzla or Fazil Akhlat* (excess or waste humors). These waste materials can accumulate in the body, the healthy organs transfer this burden to weaker tissues or organs. Joints and bones that are weak due to some issues become prime sites for such accumulation. Leg joints get mostly affected by it due to their anatomical structure and position ^[12, 13]. Factors for formation of *fuzla*, includes

overeating, alcohol consumption, poor digestion, lack of physical activity, excessive sexual activity, emotional stress, and digestive issues like flatulence [7, 8, 10-13, 24].

According to the ancient physician *Qamri*, the disease arises from waste matter which are expelled by the vital organs (*A'aza-i-Raisa*) and tend to settle in the peripheral parts of the body, particularly where there is weakness in the joint. These harmful substances are believed to be generated primarily due to chronic digestive issues (dyspepsia) and liver dysfunction [24].

Hippocrates was among the earliest to describe gout as a condition resulting from the excessive buildup of one of the body's four humors most commonly phlegm. He believed that this excess humor would collect in the joints, causing painful swelling. He demonstrated that overeating, sexual excess, together with a sedentary lifestyle linked to this condition. Later, *Galen*, added a hereditary factor and observed that the disease inherited individual often experienced more severe symptoms than those who acquired it through lifestyle changes [5].

Ibn Hubal Baghdadi attributed gout to either hot, cold, or more often, a combination of multiple humors (*murakkab madda*). The accumulation in the joints occurs due to imbalance either in *Dam* (Blood), *Balgham* (phlegm), *Safra* (yellow bile), or *Sawda* (black bile), leading to pain and inflammation [12].

Zakariyya Razi explained, *Niqris* as the build-up of kaimoos in the joints. He elaborated that the accumulation stretches the surrounding nerves, leading to pain. Razi identified primary cause (*madda-e-marz*) is often *dam* (Blood), though in many cases, it was due to *balgham* (phlegm) or a mixture of *balgham* and *safra* (phlegm and yellow bile). He noted that when phlegm thickens especially when mixed with other substances it can resemble pus. If this mixture stays in one place for too long, it becomes more concentrated and begins to harden. According to Razi, if the accumulated matter is thin, it may be resolved quickly; however, if it is thick, the condition becomes more stubborn and takes longer to clear [11].

Ibn Sina also discussed *Niqris* in terms of humoral imbalances. He noted that the matter could vary it might be pure blood (*dam*), or a combination such as blood and phlegm, blood and yellow bile (*safra*), or blood and black bile (*sawda*). In some cases, pure phlegm, or a mixture of phlegm and yellow bile (*balgham murra*) could be a sole cause [10].

Azam Khan notes that when this matter is thick and dense, the pain tends to be stronger, inflammation becomes more noticeable, and the duration of symptoms is prolonged. But when the matter is thin the painful episodes can subside soon.⁷ According to Hippocrates, *Galen* *Niqris* tends to appear more frequently in *Rabi* (spring) and *Kharif* (autumn), with symptoms worsening in the *Kharif* season, especially in individuals who consume a lot of fruits during this time [11].

Most of ancient physicians agree that *Balgham-e-Murrah* (bitter phlegm) is the primary cause. After it the most is phlegm such as *Balgham Kham* (Pure phlegm) or *Balgham Jassi* (thick phlegm), then it can be *Dam* (Blood) then *Safra* (Yellow bile) can sometimes contribute to the condition, although very rare for pure *Sawda* (Black bile) to be a cause [10, 11, 14].

• Modern Medicine

Clinical Features

Acute gout attack can be a sudden and severe episode that demonstrates all the stereotypical signs of acute inflammation. The affected joint is very painful, swollen and tender and the skin over the joint looks hot, red and dry in contrast to the moist skin that is commonly witnessed in cases of septic conditions. Systemic manifestations of this local inflammation may include fever, increased white blood cell count (especially neutrophils), and raised acute-phase reactants, although some people may experience only a single episode of gout, others may develop chronic tophaceous gout or even kidney stones (nephrolithiasis) without necessarily having repeat acute exacerbations. The metatarsophalangeal joint (joint of the big toe) is mostly affected. But over time other joints such as the knees, ankles, wrists and little joints of the hands and feet will also be included. Approximately 15 percent of patients can exhibit a polyarticular (multiple joint) pattern.

The symptoms are different in chronic tophaceous gout. Patients usually complain of a dull, constant ache rather than acute attacks which are sharp and painful. There is also joint swelling and stiffness. Small deposits of urate crystals, known as tophi, are typical of chronic gout and may appear in places with limited blood supply, including the outer ear, along tendons and joints or the extensor surface of the limbs. They are also very rare but may occur in internal organs. These deposits can push against the surrounding tissues or simply be problematic because of their position. In others, they can cause the skin to break releasing a chalky toothpaste-like substance and creating a permanent sinus. Wounds caused by spontaneous rupture or surgical excision of tophi can take a long period to heal [25].

• Unani Medicine

Clinical Features: The sign and symptoms depend upon the type of *Madda* (matter) accumulates in the joints. When the madda is *Damwi* (blood), the joint will be hot, red with tightening of joint. When it is *Safrawi* (yellow bile) then the affected site will be pale yellowish, very hot with minimal redness with mild to moderate pain and tightening of joint, but swelling will be minimal. Due to excess of *Balghami* (phlegm) on joint, the colour of the skin is normal or less bluish with less burning sensation and persistent pain. When *Madda* is *Sawdawi* (Black bile) the site is black or green in colour, lacks elasticity with minimal inflammation and mild pain. A part from this all other sign and symptoms related to the humor can be present [10, 11, 23].

Classification of Gout in Unani Classical Literature:

Unani physicians have classified gout (*Niqris*) based on the nature of the disease, involvement of organs, and severity of symptoms. The classification is according to dominant humors (*Akhlaat*), severity, and affected areas.

- **Based on Humors (*Akhlaat*):** Single Humoral Dominance-*Damwi* (Blood), *Safrawi* (Yellow bile), *Balghami* (Phlegma), *Saudawi* (Black bile) and Combined Humoral Dominance [10, 13, 14].

Qustha Ibn Luqa classified gout based on the type of accumulated waste (*Fuzulath*) or humour:

- ***Murrah Safra*:** Related to bilious humour (*Safra*) and ***Balgham Ghaleez*:** Related to thick phlegmatic humour (*Balgham*) [26].

Samarqandi provided a more detailed classification according to severity and clinical features.

- **Haad (Acute), Muzmin (Chronic), Nigris Muffasali (Articular):** Gout affecting joints. *Nigris Hashvi* (Visceral): Gout involving internal organs [8].
Razi and *Rabban Tabri* classified gout according to its severity:
Haar (Acute) and *Barid* (Chronic) [11, 27].

Diagnosis

• Modern diagnostic tool

Biochemical analysis such as, Serum Uric Acid, Renal function tests, Liver function test, glucose and lipid profile because of association with metabolic syndrome, Measurement of Hypoxanthine-guanine Phosphoribosyltransferase (HGPRT) and Phosphoribosyltransferase (PRPP) levels, Synovial fluid analysis, Joint X-rays Chemical analysis of renal stone (If renal stones suspected) [17, 28].

• Unani diagnostic tool

Ibn Sina explains that the matter can be identified according to the site colour or by touching the site (hot or cold) or by urine and stool [10]. *Razi* explains that the nature of the accumulated matter influences the disease outcome. If the *Madda* is thick and sticky, the prognosis is usually poor. On the other hand, if it is thin and light, the chances of recovery improve. When both thick and thin forms of *Madda* are present together, recovery is slower but generally occurs within 40 days if proper management is maintained [11].

Management

• Modern medicine

Oral colchicine or NSAID; Glucocorticoids

- **Xanthine oxidase inhibitors:** Allopurinol, Febuxostat, Uricosuric drugs: Probenecid, Sulfinpyrazone, Benzbromarone.

Lifestyle measures are as important as drug therapy in treatment of gout. Patient should be advised to lose weight, avoid high purine diet, alcohol and certain type of medicine which can increase gout attacks [17, 28].

Surgical interventions

• Unani medicine

Principles of treatment (*Usool-e-ilaj*) or *Ahkam Nigris*:

In treatment of *Nigris* the following should be done:-

- Strict restrictions which alleviate *nigris*.
- Restrictions in Food and drinks.
- Treatment through purgatives (*mushil*).
- Treatment through vomitings (*Qai*).
- Treatment through venesection (*Fasad*).
- Nutool on both feet.
- Local application (*Tila wo Zimad*) Local application.
- Treatment through bathing (*Hammam*).
- After cure and healing from disease prevention should be taken, so that the disease should not come back.
- Treatment should be stored early so that the disease cannot grow [26].

Treatment

After identifying the *madda nigris* (matter of *nigris*) treatment is done according to it.

According to *Maseehi* if *Madda Nigris* is *Damvi* (Bloody)

try to decrease it by *Amal-e-Fasad* (venesection).

If it is *safravi* (yellow bile) then treat it by *Mushil* laxatives and purgatives.

If the *madda* is *balghami* (phlegm) do *tanqiya* (evacuation) by emetics [17].

According to the humour involved *Mundij* (Concoctive) and *Mushil* (Purgative) is given. After evacuation *Muaddal* (Alterative), *Muhallil* (Anti-inflammatory), *Musakkin* (Analgesics) should be given [26].

In treatment of *Nigris* medicine having the following properties are given:-

- *Mundij* (Concoctive), *Mulayyin* (Laxatives), *Mushil* (Purgatives), *Mulattif* (Demulcent), *Mudirr-e-Bawl* (Diuretic), *Musakkin-i-alam* (Analgesic), *Muhallil-e-auram* (Anti-inflammatory).
- Localized fomentation [26].

Dietotherapy

Eat less food and easily digestible food like chapatti, spinach, fenugreek, rice boiled in milk, pear etc.

Avoid chickpeas, mint, radish, cucumber, onion, milk and dairy products, fish, meat, alcohol which cannot be easily digested

Avoid food according to the type of humour involved which is linked and can increase the *madda* of *nigris*.

Avoid immediate bath and exercise after food [8, 11, 26].

Regimental therapy

Before initiating any treatment, it is essential to consider several factors related to the patient's lifestyle, physical condition and body's temperament. Treatment approaches based on the type of *Madda* (morbid matter) involved. In cases where the imbalance is due to *Dam* (blood), *Fasad* (venesection) is done followed by purgation. After this the patient should be given *Maul buqool* and the drinks which calm the heat of blood. This should be done in the season of *Mavsam-Rabi* (spring) which is considered very beneficial to the patient. After this exercise should be advised with diet restrictions [11]. For venesection of foot the *Warid Basaliq* (basilic vein) on the same side is chosen and for venesection of hand the opposite side is selected [24]. *Ibn Zohr* advised venesection on *Warid Qifal* (cephalic vein) of opposite side. Hippocrates suggested venesection of *Warid Safin* (Saphenous vein) is more beneficial. The amount of blood withdrawn depends on the patient's strength, season and temperament [26].

Douche (*Nutool*) is done according to the time, severity of disease and type of *madda* involved. Bath (*Hammam*) is useful at the end of disease when the *madda* is very less which can be dissolved and excreted through sweat. The water for bath should be sweet and normal temperature. Simple exercise should be done and severe exercise is prohibited [26].

Pharmacotherapy

According to Unani scholars, first root cause should be determined, then proper steps should be taken to eliminate the harmful substances (morbid matter) from the body using various methods, such as purgatives, diuretics, and diaphoretics [8]. When there is an excess of *Balgham* (phlegm), emetic should be given then *Mundij wo Mushil-i-balgham* (Phlegm Concoctive and Phlegmagogue) should be given [26]. When excess of hot *Safra* (yellow bile), occurs

physicians are advised not to rush to cool down (*Tabrid*) as it can cause matter to spread to vital organs, which may lead to severe complications even death ^[7]. To manage this safely, emetics should be administered first then *Mundij-i-Safra* wo *Mushil-e-safra* (Bilious Concoctive and Cholagogue) should be done ^[14]. Though *sauda* (Black bile) is rare but if occurs evacuation should be done by using *Aftmoon* (*Cuscuta reflexa*), *Bisfaij* (*Polypodium vulgare*, Linn) ^[7].

If the matter is *murakkab* (mixed humors) it is very difficult for the treatment so the medicines which have the property of eliminating more than one humours should be used ^[26]. In case of phlegm and yellow bile mixture *Suranjan* is very beneficial that can be given with some other medicine which decrease its side effects and increase its efficacy ^[14].

After evacuation of *madda* (morbid matter) completely *Muaddal* (Alterative), *Muhallil* (Antiinflammatory), *Musakkin* (Analgesics) should be given. Jalinus explains *Mulattif* (Demulcent) medicines along with *Mudirr-i-bawl* (diuretics) such as [*Zarawand Madhiraj* (*Aristolochia rotunda*, Linn), *Sudab* (*Ruta graveolens*)] are extremely useful. *Razi* further elaborated that he noticed that many Unani scholars agree that the use of diuretics are very useful in the disease *Niqris*. In addition to oral medications, topical treatments are also recommended ^[26].

Restrictions and Preventions

Eat less food, Eat easily digestible food, Avoid Alcohol, avoid coitus, avoid stress and do more exercise ^[11].

Single drugs used for NIQRIS (Gout) ^[11, 29-34].

Single Drugs	Botanical Names	Parts Used
<i>Suranjan</i>	<i>Colchicum luteum</i>	Corm
<i>Bozidan</i>	<i>Tanacetum umbelliferum</i>	Roots
Shahm Hanzal	<i>Citrullus colocynthis</i> Schrad	Pulp
<i>Tukm karafas</i>	<i>Apium graveolens</i> Linn	Seeds
<i>Haldi</i>	<i>Curcuma Longa</i> Linn	Rhizome
<i>Zarawand</i>	<i>Aristolochia rotunda</i>	Roots
<i>Sudab</i>	<i>Ruta graveolens</i> Linn	Leaves, Seeds
<i>Maida lakdi</i>	<i>Litsea glutinosa</i>	Barks
<i>Peepili</i>	<i>Piper longum</i> Linn	Fruits, Roots
<i>Sana</i>	<i>Cassia augustifolia</i> Vahl	Leaves

Compound formations

Habb-e-Suranjan, *Habb-e-Niqris*, *Habb-e-Abufazl*, *Habb-e-Bar-ul-saata*, *Habb-e-Mtan kabir*, *Sufoof-e-Mudir*, *Jawarish-e-zarroni ambary banuskha kala* ^[35].

Majoon-e-Niqras, *Majoon-e-Yahya bin Khalid*, *Jawarish-e-zarroni ambari* ^[36].

Habb-e-Astam khiqoon, *Habb-e-Baryuma*, *Raughan-e-Naf-e-warm-e-niqras*, *Zimad-e-waja-ul-mafasil* ^[37].

Sufoof-e-Chobchini, *Sufoof-e-Suranjan zafrani*, *Iyarij-e-Loghaziya*, *Raughan-e- Malkangani*, *Raughan-e-Suranjan*, *Raughan-e-Surkh* ^[38].

Habb-e-Leemun, *Kushta Gaudanti*, *Majun Suranjan* ^[39] *Safoof Suranjan*, *Aujaia* ^[40].

Comparative Insights

• Role of Diet and Lifestyle

- **Modern medicine:** Risk factors include purine-based diet, alcohol, overweight and lack of exercise.
- **Unani medicine:** Excessive eating, heavy/purine foods, alcoholism and lack of exercise leads to *Niqris*.

The two systems emphasize on dietary and lifestyle change in prevention and management.

Etiology-Joint Deposition

- **Modern:** Uric crystals accumulate in joints where they cause inflammation leading to pain and swelling.
- **Unani:** Accumulation of *mawad-e-fasida* / *ghaleez akhlat* in joints results in pain and swelling.

These two shows the pathological presence of the unwanted substances in the joints

Clinical Features

- **Modern:** Acute intense pain of metatarsophalangeal joint showing redness, swelling, warmth.
- **Unani:** Classical description of the pain, redness, heat, swelling of the extremities (*podagra*).

There is virtually complete overlap between symptomatology

Chronic Progression

- **Modern:** Chronic gout → recurrent attacks, tophi.
- **Unani:** Chronic *Niqris*-chronic pain, deformities of the joints, systemic spread.

Both acknowledge acute and chronic stages.

Importance of Prevention

- **Modern:** Recurrence prevented by weight control, low purine diet, restriction of alcohol.
- **Unani:** Preventive (*hifzan-e-sehat*) with diet control, physical activity, and lifestyle control eliminates relapse.

There is a focus on prevention in both

Use of Colchicum (*Suranjan*)

- **Modern:** Colchicine from(*Colchicum luteum*) is used.
- **Unani:** *Suranjan Shirin* formulations (e.g., *Majoon Suranjan*) are very common.

Similarity of pharmacology found in other context

• Identification of Co-morbidities

- **Modern:** Gout related to renal disease, cardiovascular risk, metabolic syndrome.
- **Unani:** *Niqris* is linked to metabolic disorder and systemic imbalance caused by derangement of the humors.

The two systems recognizes systemic involvement other than joints.

Conclusion

In short both Modern and Unani systems have overlapping views on Gout (*Niqris*) as a diet-related, lifestyles-related, metabolic-imbalance-related, deposition in joints, and acute chronic and long-term management disorder.

Conflict of Interest

Not available

Financial Support

Not available

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How to Cite This Article

Begum A, Begum Z, Rasheeda AM. Niqris (Gout): A comprehensive review of concepts and management in modern and Unani medicine. International Journal of Unani and Integrative Medicine. 2025;9(2):277-283.

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