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A Review of Gout in Unani and Modern Medicine: Correlating Gout and *Niqris*

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Abstract

Gout, known as "*Niqris*" in Unani medicine, is derived from the Unani word "*Anqarūs*," meaning the joint of the great toe. It typically occurs in the great toe, thus named *Niqris*. Historical descriptions by Ibn Sina (980-1037 AD) and Ibn Nafees (1210-1288 AD) detail that Niqris pain often starts from the great toe but can also begin at the heel or ankle and spread across the foot, sometimes presenting with redness, tenderness, swelling, rigor, and low-grade fever. In Unani literature it is classified as *Damwi* (Sanguineous) *Safrawi* (Bilious) *Balghami* (Phlegmatic) and *Saudavi* (Melancholic) based on humours (*Akhlaat*) involved. In modern medicine, gout is caused by the deposition of monosodium urate crystals in joints and tissues. Risk factors include male sex, obesity, hypertension, alcohol intake, diuretic use, diets high in meat, seafood, and fructose-rich foods and beverages, chronic kidney disease, certain ethnic groups, and living in high-income countries. Clinically, gout is characterized by swelling, pain, or tenderness in peripheral joints or bursae, often with tophus development. Diagnosis can be made using validated clinical prediction rules, and arthrocentesis with synovial fluid or tophus. Acute gout episodes are treated with colchicine, NSAIDs, and corticosteroids. Although modern treatments are effective, they often have adverse effects, leading to increased interest in traditional systems of medicine like Unani. In the Unani System of Medicine, it can be managed by using regimental therapy, diet therapy and pharmacotherapy.

Keywords: *Niqris*, Gout, Unani Medicine

Literature Review

Historical Background

Gout is among the oldest known diseases, identified around 2640 BC when Egyptians first documented Podagra (foot pain) or gouty arthritis, typically affecting the big toe. Today, it is recognized as uric acid arthropathy. [1]

The earliest mention of gout is in the Ebers Papyrus (1500 BC), which describes a drug likely corresponding to colchicine.[2]

Hippocrates (Buqrat, 460 – 377 BC), also known as the "father of medicine," lived in the fifth century and is credited with accurately describing the condition. He called it "*the unwalkable disease,"* a condition that primarily affected older men and was a result of living a luxurious lifestyle. Those who experienced it could not walk due to the extreme discomfort.[3]

The name "Gout" was coined by a monk named *Randolphus* of Bucking in the 13th century from the Latin word "*Guuta*" (which translates into "Drop").

From the 17th to 18th century, significant advancements were made in understanding gout. Antoni van Leeuwenhoek described the microscopic structure of tophi in 1679, while Thomas Sydenham provided detailed symptomatology of acute gout in 1683. By the mid-18th century, William Stukeley and Scheele identified uric acid crystals in tophaceous joints, and colchicine was rediscovered in 1763. Wollaston confirmed tophi as uric acid deposits in 1797, and a century later, McCarty and Hollander used polarized microscopy to detect monosodium urate crystals in joint fluid. [4]

In the 19th century, Sir Albert Baring Garrod hypothesized that urate deposits caused gout and recommended a low-purine diet. Experiments by Haig and Wailer supported this dietary approach. By the early 20th century, gout was recognized as a metabolic disorder, with treatments like aspirin, probenecid, and allopurinol emerging.[5]

In the 21st century, gout remains the most common inflammatory arthritis in men over 40, with increasing incidence due to dietary changes, longer lifespans, renal impairment, and medication use. Gout is linked to higher risks of metabolic syndrome, diabetes, cardiovascular diseases, and increased mortality from coronary heart disease. Consumption of fructose-rich drinks has also been associated with higher gout risk.[6,7,8,9]

Gout was first explained radiologically by Huber in 1896. Its pathogenic mechanism was provided in 1898 by Emil Fischer.[10]

Introduction

Niqris (gout) is well known in the Unani system of medicine also; according to *Ibn-e-Habal* "*Niqris*" originated from the word "*Anquroon*" which means big toe of the foot, because this disease usually starts with the involvement of this particular joint and hence the disease has been named after this joint.[11,12,13]

According to *Ibn Hubal*, The word Niqris is derived from the term '*Naqoroos*,' which indicates 'the joint of the great toe.' Because this disease classically affects the first metatarsophalangeal joint, this name has been given.[14]

According to *Ibn e Sina*, "*Niqris* is a type of arthritis that can start with the toes, particularly the great toe, the ankles, under the foot, or sometimes it starts from one side of the foot, affects the entire foot, and then rises up to the thigh." Inflammation can occasionally affect nerves, ligaments, and tendons in addition to the surrounding tissue of the joints.[15]

Ismail Jarjani has described that as *Mavad-e-fazooni* (morbid humours), which accumulates in the small joints and tendon if it causes pain and inflammation in small joints called *Niqris*. It occurs mainly in the greater toe. The ankle joint and the joint of the toes may also be involved.[16]

According to *Ali-Ibn-Abbas-Al Majoosi* (930-994), the discomfort that can occur in the joints of one or both legs, as well as occasionally in the wrist or elbow, and mostly in the big toe joints, is referred to as *Niqris*.[17]

Classification of Gout in Unani Classical Literature

Unani Physicians are classified as Niqris according to the conditions of the disease, involvement of organs, and severity of symptoms.
Based on humours (*Akhlaat*) involved, it is classified as Single humours dominance: Damwi, Safrawi, Balghami and Saudawi &
Combined humours dominance: *Dam-e-Safrawi, Dam-e-Balghami.* [18]
Qustha Ibn Luqa has described two types of *Niqris* according to accumulated *fuzulath* (*Khilt*):
One of them related to *Safra* that is "*murrah-safra*"
Another one is related to *Balgham,* that is, "*Balgham Ghaleez.* [19]

Rabban Tabri and Rhazi classified it based on the severity as *Haar* (Acute), and *Barid* (Chronic)[20].

Samarqandi classified *Niqris* as – *Haad* (Acute), *Muzmin* (Chronic), *Niqris mufassali* (articular), and *Niqris hashvi* (visceral).[21]

Hakeem Mohd Hasan Qurshi has classified gout into three main types namely- *Haar* (acute), *Barid* (chronic) and *Murakkab* (combined).[22]

According to Hakeem Ghulam Jeelani, gout is classified into four main types- *Niqris shadeed* (acute), *Niqris purana* (chronic), *Niqris andruni* (visceral), and *Niqris beqaida* (irregular).[23]

Hakeem Ghulam Jeelani in *Makhzanul Jawahar* has classified according to involvement of organ as *Niqris-e-Haar* (Acute Gout), *Niqris-e-Baarid* (Chronic Gout), *Niqris-e-Hashwi* (Visceral Gout), *Niqris-u-Raas* (Cephalagra), *Niqris-u-Rukba* (Gonagra), *Niqris-u-Sullamiyat* (Phalnjagra), *Niqris-ul-Qadam* (Podagra), *Niqris-ul-Qalb* (Cardiagra), *Niqris-ul-Kataf* (Omagra), *Niqris-ul-Yad* (Cheriagra), *Niqris-ul-Waraq* (Ischiagra), *Niqris-e-Mafasili/ Muntazim* (Regular or Articular Gout), *Niqris-e-Muntaqil / Munqata* (Retrocedent or Suppressed Gout).[24]

The pathophysiology of *Niqris* in Unani literature

The food substances absorbed by *Quwat-e-jazeba* and retained by *Quwat-e-maseka* but not yet acted upon by *Quwat-e-Mughaiyyera* to be properly absorbed by the digestive system and become part of the body instead turn into toxic substances known as *Fazil Akhlat*. [25] These toxic substances, or *Fazil Akhlat*, can deposit anywhere in the body and contribute to the pathophysiology of gout. This *Akhlat Fazila* can be of *Damvi, Safravi, Balghami, Saudavi* types, or a combination of any two, as stated by Sheikh in his book Al Qanoon Fit-Tib. According to *Ibn Sina*, the Madda of *Niqris* can be pure *Dam*, a combination of *Damvi-Balghami, Damvi-Safravi, Damwi-Saudawi,* or *pure Balghami*, and it can also be *Balgham-e-Murra*. Most ancient physicians agree that *Balgham-e-Murra* is the main cause *of Madda-e-Niqris,* followed by pure *Balgham, Dam, and Safra*. Rarely is the cause of Madda-e-Niqris pure *Sauda*.[26]
Allama Qarshi explained that pure *Balgham,* due to its consistency, cannot penetrate and remain in the joints unless it is mixed with *Balghami-e-Murra,* which alters the consistency of pure *Balgham,* making it more easily penetrable into the joints and thus causing gout. Regarding *Balgham-e-Kham,* he wrote that it is commonly associated with joint pain. Blood can enter the joint either through a vessel or by other means, but since vessels are fewer near the joints, *Madda-e-Niqris* will not spread extensively, making *Khilt-e-Dam* a less likely cause of gout. *Khilt-e-Safra,* due to its hot temperature and high penetrative power, infiltrates nearby organs and thus cannot reach the joints and, therefore, cannot cause gout. Similarly, *Khilt-e-Sauda,* due to its consistency, cannot penetrate the joints and thus cannot cause gout. *Ismail Jarjani and Azam Khan* agree with *Ibn Sina's* philosophy. [27]

Zakariya Razi presented a new perspective on gout. He stated that *Balgham-e-Kham*, which is somewhat concentrated and resembles pus, is the cause of gout. He wrote that the disease (Madda-e-Marz) is often *Dam*, but most frequently, it is *Balgham-e-Safravi*. *Balgham* is a type of humor that becomes more concentrated and pus-like when mixed with raw matter. When it remains in one place for some time, it becomes more concentrated and solidified.[28]…1 W

Clinical manifestation of *Niqris* (Signs and Symptoms)

Unani scholars mentioned the clinical manifestation of *Niqris* according to its cause.

Su'al Mizaj Sazij (Simple Imbalance of Temperament)

This rarely occurs, but when it does, it goes away quickly. The dissolution of Su'al Mizaj Sazij haar is happening faster. This type does not exhibit swelling or indicators of completion, and the patient feels relieved when using items with opposing temperaments.[29]

Su'al Mizaj Maddi Mufrad (Single Humoral Imbalance)

When the cause of the issue is sanguine (Dam), the skin over the affected area is red. There is noticeable swelling accompanied by pain and tenderness, and patients find relief through venesection (Fasd).In cases involving *Safrawi* (bilious) matter, swelling is rare. There will be severe aching and signs of inflammation in the affected area, along with other symptoms of bilious matter in the body. If the problem is due to phlegmatic *(Balghami*) matter, the skin over the affected area may be normal, pale, or white. Swelling is *soft*, with persistent low-grade pain and a lack of warmth being typical features. Patients benefit from warmth, while cold exacerbates their condition. The melancholic type's skin over the affected area is dry, lacking laxity, luster, and warmth. The pain is mild, the swelling is hard, and the skin color is slightly black or bluish.

Su'al Mizaj Maddi Murakkab (Combined Humoral Imbalance)

If the patient's condition is due to a mutual impairment of the blood and yellow bile *(Dam and Safra*), they appear young, have prominent blood vessels on their face, enjoy spicy and hot food, and have scarlet urine. Hot items aggravate symptoms, cold items alleviate symptoms, and venesection also relieves symptoms. When it is brought on by phlegm and blood impairment combined (*Balgham and Dam*), the patient is usually elderly, dark-skinned, obese, and has a slow rate of activity. Sedentary behavior, excessive water consumption, excessive salivation, using *Hammas* (Turkish baths), engaging in sexual activity right after eating, and excessive cold intake are all past experiences. The patient has more viscosity and white urine.[30]

Gout

Gout is defined as an inflammatory crystal-induced arthropathy that affects peripheral joints**.**42 It is a disease of both metabolism and inflammation.[31]

It is a disorder in which plasma uric acid is elevated, and urate crystals precipitate in the joints, causing pain, inflammation and swelling of the affected joints (often in the big toe)
Gout (arthritis urica) is inflammation of joints caused by the deposit of mononatrium-urate-monohydrate (MNU) crystals in the tissues.[32]

Gout is the most common form of inflammatory arthritis globally. The risk of gout increases with age, and it is thus more common in aging populations.[33]

The prevalence of gout ranged from 1 - 4% worldwide, and incidence ranged from 0.1-0.3%.3 - 4 times more common than rheumatoid arthritis. The prevalence of gout increases with advancing age, a pattern that is seen over the entire lifespan in men and especially after menopause in women, possibly owing to the uricosuric effects of estrogen.[34]
Gout is more common in men vs. women by 3:1 to 10:1.70. In Western countries, it occurs in 3–6% in men and 1–2% in women.[35]

Hyperuricemia and Gout: Causes and Pathogenesis

Biologically significant hyperuricemia occurs when serum urate levels exceed solubility (~6.8 mg/dL). While common, hyperuricemia does not always lead to gout. Humans produce 250 to 750 mg of uric acid daily from dietary purines and the breakdown of dying tissues. The exact cause of gout is unknown but may involve genetic defects in purine metabolism. Uric acid, a trioxypurine with three oxygen groups, is the most insoluble purine substance. Gout pathogenesis begins with urate crystallization in joints, bursa, or tendon sheaths, leading to inflammation from phagocytosis of monosodium urate (MSU) crystals, typically associated with elevated blood uric acid levels.

Uric acid, derived from purines like adenine, guanine, hypoxanthine, and xanthine, results from purine metabolism. Adenine and guanine are found in DNA and RNA, while hypoxanthine and xanthine are intermediates in purine nucleotide synthesis and degradation. Both undissociated uric acid and MSU are sparingly soluble.

The body's urate levels depend on dietary intake, synthesis, and excretion. In primary gout, defects in purine metabolism cause hyperuricemia, resulting from increased uric acid production or abnormal retention. Hyperuricemia in gout patients results from overproduction in 10% and underexcretion in 90% of cases. Overproduction is often due to increased cell turnover from proliferative and inflammatory disorders, pharmacologic interventions, or tissue hypoxia.

The kidneys handle urate through glomerular filtration followed by partial tubular reabsorption, with only about 20% of filtered uric acid being excreted. Elevated uric acid levels can predict renal failure in patients with pre-existing renal disease and cause interstitial and glomerular changes similar to chronic hypertensive changes. Hyperuricemia is also epidemiologically linked to hypertension and may independently contribute to its development.[36,37,38]

Hyperuricemia and gout may be classified as follows.[31]
Primary: (90%) These cases seem to be innate—that is, they are neither secondary to an acquired disorder nor the result of a subordinate manifestation of an inborn error that leads initially to a major disease, unlike gout. Some, but not all, primary gout cases have a genetic basis.
Secondary: These cases develop in the course of another disease or as a consequence of drug use.
Idiopathic: In these cases, a more precise classification cannot be assigned.

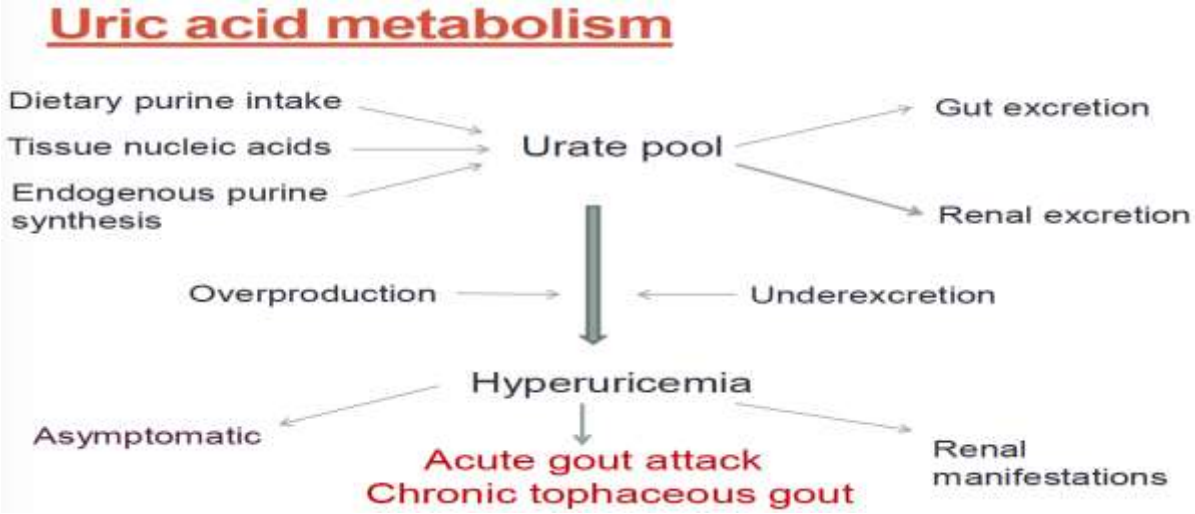

Fig: Uric Acid Metabolism

Clinical Presentation

Hyperuricemia and gout progress through four phases:[39,40]

Asymptomatic Hyperuricaemia: Elevated uric acid levels are often detected incidentally and might never result in gout. Acute urate overproduction, such as during cytotoxic chemotherapy, can cause severe renal failure. This stage requires close monitoring but no active treatment.

Acute Gouty Arthritis: Commonly affects the big toe (first metatarsophalangeal joint) but can also involve other joints like the instep, ankle, knee, or hand. An acute attack often starts at night, presenting with swollen, hot, red, shiny, and extremely painful joints, sometimes accompanied by mild fever and chills. Untreated attacks may improve within one to two weeks, with possible skin peeling over the joint. Atypical forms include tenosynovitis, bursitis, cellulitis, or mild discomfort without significant swelling. Acute gout in one joint can lead to migratory attacks in other joints (cluster attacks), with women, particularly those on diuretics, experiencing more polyarticular attacks.

Intercritical Gout: After the initial gout attack, subsequent episodes may be infrequent or occur after several years. However, most patients experience additional attacks within a year, with increasing frequency and involvement of more joints over time.

Chronic Gout (Tophaceous Gout): This stage develops after prolonged, uncontrolled hyperuricemia, usually following at least ten years of gout.

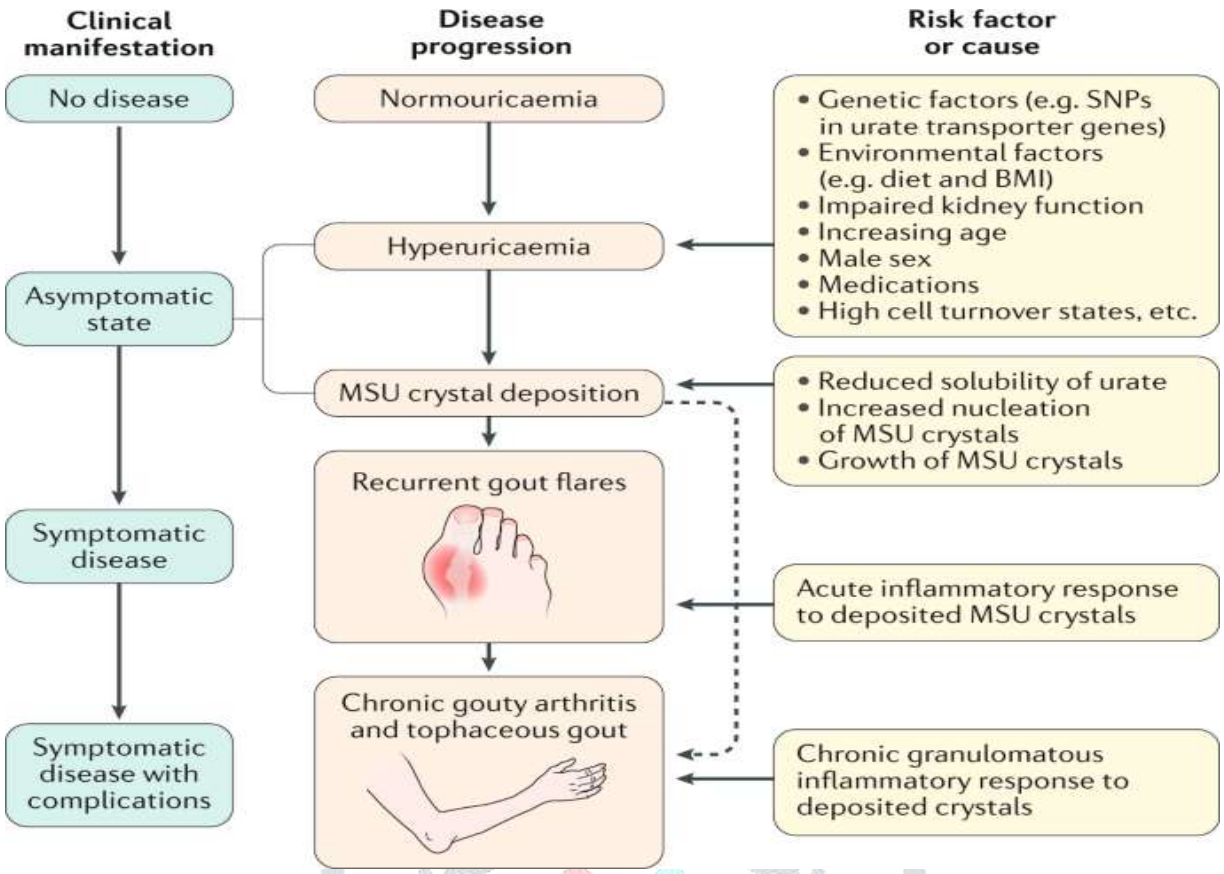

Fig: Progression of Gout

Management

Allopathic Treatment

Allopurinol is the preferred initial treatment for hyperuricemia, with dosage adjusted to achieve optimal serum (<6 mg/dL) or 24-hour urine (<600 mg/day) uric acid levels. If ineffective or not tolerated, febuxostat is recommended, especially if there is a genetic risk of allopurinol hypersensitivity syndrome.

Probenecid, with or without colchicine, can be used for gout prophylaxis but is not recommended for patients with uric acid nephrolithiasis or hyperuricosuria (>800 mg urinary uric acid/24 hours).

Pegloticase is used for intractable, symptomatic hyperuricemia when other treatments are ineffective or intolerable.

Rasburicase is recommended for temporary use in acute.

Unani usool-e-ilaj wa ilaj(Line of Treatment in Unani)

Izale sabab (Elimation of the causes)

Ilaj bil ghiza (Dieto therapy)

Ilaj bid tadbeer (Regimental therapy)

Ilaj bid dawa (Pharmacotherapy)

Ilaj bil ghiza (Dieto therapy)

1. Avoid red meat, high sugar intake, and alcohol consumption.
2. Steer clear of high-purine diets.
3. Do not eat immediately after coitus or a Hammam (bath).
4. Eat meals in a calm and peaceful environment.
5. Consume foods that help thin the blood (Muraqqiq-i-dam).
6. Opt for easy-to-digest foods (Zud Hazm ghiza) such as roti, milk, rice, barley water (daliya), spinach, fenugreek, tomato, pear, and apple juice.

Ilaj bid Tadbeer (Regimental Therapy)

Regimental therapy is a widely popular treatment method that uses physical approaches to enhance the body constitution by eliminating morbid or waste materials. This therapy improves health quality by boosting the body's defense mechanisms.

Ilaj bid dawa (Pharmacotherapy)

Many single and compound drugs which are the very effective in Gout 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*), Suranjan (*Cholchicum luteum*). Compound drugs: Habb-e-leemun, Habb-e-Suranjan, Jawarish Zar" uni Ambarin Ba Nuskha Kalan, Majun Suranjan, Bonigra capsule, Rogha-e-Shifa, Roghan-e- Surkh, Roghan-e- Mufasil.[42]

Discussion and Conclusion

From the detailed literature on both Unani and modern aspects of gout, we can correlate gout with Niqris based on the following points:

In modern medicine, gout is caused by the accumulation of uric acid crystals, while in Unani medicine, it is primarily due to the accumulation of humors (Dam, Balgham, Sauda, Safra, or a combination). Thus, both perspectives identify the cause as the deposition or excess of material or madda.

Gout is common in people who consume high-protein diets or wine. Hence, it is known as "the disease of kings."

It may remain asymptomatic or present as an acute attack.

Gout often starts as an acute episode in the great toe.

There is a close resemblance in the signs and symptoms of gout and niqris.

Modern and Unani medicine management includes sedatives or anti-inflammatory drugs, removal and inhibition of deposition of morbid substances, i.e., *Mavad-e-fazooni* or uric acid crystals, and dietary protein restriction.

Colchicine is the key drug used in modern medicine, while in Unani medicine, Suranjan, which also contains colchicine, is primarily used for managing Niqris.

Hence, we can conclude that the Unani classical literature on this disease closely aligns with the modern description of gout, mainly with respect to clinical presentation.

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Conflict of interest

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