

An Overview of *Niqris* (Gout) and its Interpretation with Hyperuricemia

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Abstract There is a comprehensive description of *Niqris* in classical Unani literature. Ancient Greco-Arabic scholars have described *Niqris* in detail along with its clinical features, Etiopathogenesis, complications and management in their treatises. According to Ibn Sina *Niqris* is a type of pain which sometimes starts from fingers, toe and sometimes from heel. In unani system of medicine the term hyperuricemia as such is not described at all, but a disease with the name of *Niqris* has been mentioned in most of the classical text, whose clinical presentation and causes very much resembles with the gout (which occur due to hyperuricemia). Gout is a common metabolic disorder, typically presenting as an acute monoarthritis, most commonly of the first metatarsal phalangeal joint. The underlying problem is a build-up of urate, a purine breakdown product. Unani physicians claim to possess many safe and effective drugs for the management of *Niqris*. Therefore, it is one of the areas which have to be given priority in scientific research in Tibbe Unani.

Keywords *Niqris*; *Hyperuricemia*; *Gout*; *Unani Medicine*

1. Introduction

According to the renowned Unani physician, Buqrat (Hippocrates), *Niqris* is a joint disease which is caused due to excess of one of the four humors, which under certain circumstances, drop or flow into a joint causing pain and inflammation [1]. Gout is the term used to describe a group of disorders which results from tissue deposition of crystals of monosodium urate monohydrate from hyperuricemic body fluids. It is usually a monoarticular arthritis, although uncommonly presents as a polyarticular disease. It is characterized by intra-articular deposition of uric acid crystals. It is associated with hyperuricemia, which may be produced by thiazide diuretics [2, 3, 4].

Hyperuricemia denotes an elevated level of urate in the blood. Hyperuricemia alone is not the sole factor for the development of clinical disease. Tissue deposition of crystals of monosodium urate and resulting clinical symptoms and signs of gout usually only follow prolonged elevation of serum urate. In unani system of medicine the term hyperuricemia as such is not described at all, but a disease with

the name of *Niqris* has been mentioned in most of the classical text, whose clinical presentation and causes are very much resembles with the gout (which occur due to hyperuricemia). Gout is more common in men, the sex ratio being 20:1 and the mean age at onset 40 years; in women, the onset of gout is postmenopausal [5]. Hyperuricemia is a result of multifactor interactions including gender, age, genetic and environmental factors. Classically, the following conditions are associated with hyperuricemia: alcoholism, obesity, hypertension, dyslipidemia, hyperglycemia, diabetes mellitus, lithiasis, renal failure, and medication use (diuretics, cyclosporine, and low-dose aspirin) [6]. Owing to dreadful complications of Gout and lack of safe and effective drug for its management, it becomes a thrust area for research, in every field of medical science. The researchers of different systems of medicine are continuously concentrating themselves for the development of safe and effective drug for the management of Gout. As far as the Unani system of medicine is concerned, *Niqris* is being treated since Greco-Arab period. Unani physicians claim to possess many safe and effective drugs for the management of *Niqris*.

2. Epidemiology

In the general population, the prevalence of hyperuricemia ranges between 2 – 13.2%, and the prevalence of gout is between 1.3 and 3.7%. [7].

Prevalence of hyperuricemia varies according to the age, sex, race, geographical conditions and association with other diseases [8]. Gout becomes commoner with increasing age. In men the reported prevalence ranges from < 0.5% in those aged under 35 to over 7% in those aged over 75. It is rare in premenopausal women but increases to 2.5 – 3.0% in those aged over 75. The later age of onset in women may relate to the uricosuric effects of oestrogens [9].

3. Classification

3.1. Acute Gout

Acute monoarthritis results from an acute attack. Severe pain, erythema, and swelling are the characteristic features of the disease. The most commonly affected joint is the first metatarsophalangeal joint (podagra), followed by knee, ankle/metatarsus, wrist, and fingers. Polyarticular gout is less common but can occur, in those individuals who had repeated disease flares. The risk for gout is directly proportional with the degree of hyperuricemia. Acute gout is self-limited and symptoms typically resolve over the course of days to weeks [10].

3.2. Intercritical Gout

Patients of gout are usually asymptomatic in between sporadic episodes of acute arthritis. The management of patients with intercritical gout focuses on the prevention.

3.3. Chronic Tophaceous Gout

Large deposits of uric acid occur within joints or in the soft tissues, particularly around the pinna of ear, in chronic tophaceous gout. In these patients, there is substantial X-ray changes, calcification of urate deposits with soft tissue swelling and even erosions of phalangeal bone [8]

4. Etiology

The etiology of gout is multifactorial. There is a genetic component, but the operation of other factors justifies the inclusion of gout under the heading of acquired disorders. Etiological factors include:

- Gender (male > female);
- Family history;
- Diet (meat, alcohol);
- Socioeconomic status (high > low).

Alcohol consumption is a particularly common factor in promoting hyperuricemia by increasing urate production.

According to Unani literature, etiological factors of *Niqris* include excessive eating, sedentary life style, excessive intercourse especially just after eating food and lack of exercise [8, 9, 10, 11]. The active cause of *Niqris* is *Sue Mizaj* and *Raddi Mawad* (morbid materials). The morbid matter which reaches upto the joints may be of *Damvi*, *Safravi*, *Saudavi* or *Balghami* in nature [11]. The morbid humour may be mixed with and composed of phlegm and *mirrah* or any type of *madda* or *riyah*. The most common cause of *Niqris* is bile mixed with *phlegm* (Balgham with safra), then blood (*Dam*), and *sauda* is the rarest cause [12].

5. Pathogenesis

Niqris (Gout) is produced when the *madda* spells (falls) towards the lower extremities, expelled by the Vital Faculties (*Aazae Raisa*) of the body towards these extremities, which are not able to expel these matters [13]. In over 75% of patients who present with gout, there is a decrease in uric acid clearance by the kidney but the underlying cause of this is not known. In a few patients, there appears to be an idiopathic increase in the rate of purine synthesis leading in turn to increased uric acid production. The increased cellular turnover associated with a wide variety of different malignant disorders and other diseases is a common cause of secondary gout. The stimulus to the acute inflammatory reaction in acute gout is the deposition of monosodium urate crystals in the synovium and adjacent connective tissues of the joints [8]. Hyperuricemia are necessary for the development of gout. Crystal deposition can only occur when the serum is saturated with urate: ≥ 0.42 mmol/l [9].

6. Clinicopathological Feature

The clinicopathological features of gout are as follows:

- Males usually affected;
- Onset 40- 60 years, familial tendency;
- Acute inflammatory monoarthritis- more than one joint involved in 10%;
- Raised plasma uric acid (0.5mmol/l);
- Deposition of monosodium urate crystals in joints;
- Variable incidence of uric acid renal calculi;
- Mild intermittent proteinuria with focal interstitial nephritis;
- Untreated patients may progress to chronic gouty arthritis and renal failure [8].

7. Diseases Associated with Hyperuricemia and Gout

Most serious complication of gout is renal disease. The incidence of renal calculi in gout varies from country to country and the reason is not clear. In Western Europe it is of the order of 10% and gout should be considered in any patient who presents with renal colic.

Mild proteinuria is found in a proportion of patients but very few progresses to chronic urate nephropathy and renal failure. Urate crystal deposition in renal tubular epithelium induces cellular necrosis, chronic interstitial nephritis and fibrosis. Obesity, alcoholism, hypertension, Ischemic heart disease, various forms of hyperlipoproteinaemia and impaired glucose tolerance are associated with gout [8].

8. Usoole Ilaj (Line of Treatment) and Ilaj (Treatment) [1, 11, 12, 14]

In case of *Niqris Har Safravi*, avoid *Tabreed* i.e. application of barid material over the affected part because this may leads to diversion of *Mawaad* (Matter) towards the *Aazae Raisa* (vital faculties), and may cause death.

In case of *Niqris Damvi*, Maseehi recommend the use of all those tadbeer (regimens) by which the volume of blood can be reduced, while in case of *Niqris Safravi* he advice to use *mushile safra*, e.g. *Aloo Bukhara*, *Shahtra*, *Afsanteen*, *Sibr* and *Saqmoonnia*.

If **balghami**: Then do *tanqia* with drugs which are *mukhrije Balgham*.

For **Tabreed**: Apply *Zimad* with *vinegar* and *Roghane Gul*, or *aarade jau* and *Rogh. Gul*.

In between two *Mushil*, *Nuskhae Tabreed* should be given. In which *khameera gauzaban* or *Khameera Banafsha* should be given with *luabiat*.

For **Niqris Safravi**: To expel the *madda*, *maamoole matab Nuskha* (single dose)

Sibr 3.5 Masha, *Saqmoonnia*, 6 *Ratti*, *Gule Surkh 1 Masha*, *Suranjan safaid 1.75 Masha*. Make pills and use them.

Niqris Muzmin (Chronic Gout): Affected organ must be dipped in hot water for some time and then dip this organ into cold water gently.

Nutool: with *matbookh* (Decoction) of *Shaljam* (Turnip) is much beneficial for *Niqris Barid*.

If *madda* of *Niqris* is *Khilte Ghaleez Barid* then first know about its *Kamiat* (quantity) and *Kaifia't*.

Qai: For *Tanqiae Madda*

Huqna (Enema): *Qantaryun raqeeq*, *Zarawand taweel*, *Rogh sumbul Wa Shahad*

For **Tanqiae madda**: *Hab Muntin*, *Hab Sheetraj*, *Hab Suranjan*.

For **Tadeel Mizaj** and to Prevent **Madda** from Coming into the Joints: *Majoon Hurmas* or *Tiryaqe Kabir* is used-

If *khilth* is *Saudavi Ghaleez*, then avoid use of more *Mujaffif* drugs, instead apply *zimad* of *Khardal*, *bazr katan* and *Qataf* like drugs for *Tiskeen* and *Talayyen*, and do *tanqia* of the body with *aftimoon*, *Bisfaij* and *Kharbaq siah*.

If madda is *murakkab* then apply *murakkab tadabeer* according to it, and advice the *mushil* to drink.

Halaila zard 4.5 tola, *turbud safaid*, *Bisfaij*, *sana makki*, *shahtra* each 14 *masha*, *suranjan safaid*, *tukhm kasni*, *karafs*, *badyan*, *gule surkh* each 7 *masha* should be taken and make decoction of it 2 *sair* of water till it remains 0.5 *sair*, and add sugar to it.

Use of *Habbe suranjan* and *Habbe mahtabi* is also beneficial. *Roghane Hanzal* is also useful in *Niqris* and *Wajaul mufasil*.

Commonly used drugs are *Habbe Najah*, *Habbe Muntin*, *Ayarij Roofas*. These drugs are very useful in *Niqris* and *Sciatica*.

9. Conclusion

It may be concluded that *Niqris* is a humoral disease occurring as a result of imbalance in the quality and quantity of one of the four humors and results in severe pain and inflammation in joints. A number of drugs have been evaluated by the researchers of main stream medicine which possess potent and effective uricosuric activity; but the long term use of these drugs frequently leads to the development of side effects such as hypersensitivity reactions. Unani medicine axiomatically claims for successfully treating this disease since a long time without any unwanted effects.

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