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Management of hyperuricemia & gouty arthritis with unani murakkab advia & Hijama-bil-Shart

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Abstract
Hyperuricemia is considered to be one of the leading causes of joint stiffness and pain especially small joint most commonly affecting first metatarsophalangeal joint. This is an excess of uric acid in the blood. It is commonly associated with traditional risk factors such as dysglicemia, dyslipidemia, central obesity, gout (*Nikras*), gouty arthritis, tophi formation, abnormal blood pressure, etc. Concordantly, recent studies have revived the controversy over the role of circulating uric acid, hyperuricemia, and gout (*Nikras*) as an independent prognostic factor. In this regard, the role of Unani compound medicine including Habb-e-Asgandh, Habb-e-Suranjan, and Sharbat Bazoori Motadil in combination with and without Hijama Bil-Shart (Wet Cupping), evaluated on multiple patients with the possibility of lowering increased serum uric acid level. In this review, we will focus on controlling of hyperuricemia with the use of Unani Medicine and Hijama Bil Shart.

Keywords: Hyperuricemia, Uric Acid, gout, Hijama, etc

Introduction
Hyperuricemia is a condition of body characterized with excess of uric acid in the blood. Uric acid is the final product of purine nucleotide catabolism. In particular, purine nucleotides are derived from both endogenous and exogenous sources. Purine nucleotide synthesis can also occur through the activities of two different enzymes, catalyzing the single-step synthesis of a purine nucleotide from a purine bases substrate. During the reverse process, the intermediate breakdown product hypoxanthine can be 'salvaged' by the enzyme hypoxanthine-guanine phosphoribosyl transferase and then re-incorporated into nucleic acid. The whole pathway is tightly regulated and controlled by feedback inhibition. Uric acid is a weak acid, with an ionization constant of acid (pKa) of 5.75-10.3. At the physiological pH of 7.40 of the extracellular compartment, 99% of uric acid is in the ionized form as urate (as monosodium urate in blood and as potassium, ammonium and calcium urate in urine). In the urinary tract, where pH can fall to 5.7, acid uric formation is favored.

Deoxyribonucleotides and purine nucleotides catabolism leads to uric acid production. Hypoxanthine and xanthine are the intermediate products of this catabolism. Xanthine oxidase catalyzes the oxidation of xanthine to uric acid. Uric acid is the final oxidation product of purine catabolism, which means that it cannot be further metabolized. The kidneys excrete two-thirds of the total uric acid amount that is produced daily, while the remaining one-third is broken down by intestinal flora and excreted in the stool.

Hyperuricemia is potentially a harmful condition. It favors precipitation of uric acid crystals in joints and tissues, leading to complications such as gout, nephrolithiasis and chronic nephropathy. Increased levels of uric acid from excess purines may accumulate in side of body tissues and small joints especially toes and fingers form crystals. This may cause high uric acid levels. The state of hyperuricemia occurs when there is too much uric acid in the blood. High uric acid levels can lead to gouty arthritis. Elevated uric acid levels are also associated with health conditions such as heart disease, diabetes, and kidney disease. Normal Uric acid levels are 2.4 to 6.0 mg/dL (female) and 3.4 to 7.0 mg/dL (male). Causes of high uric acid levels can be primary (increased uric acid levels due to purine), and secondary (other disease condition). Sometimes, the body produces more uric acid than it is able to excrete.

Since, the body rids itself off of uric acid on urination. Hyperuricemia occurs when body either makes too much uric acid or is unable to excrete enough of it. It usually happens when kidneys are not eliminating it quickly enough. Excess uric acid levels in the blood can lead to the formation of crystals. Although these can form anywhere in the body, they tend to form

In and around the joints especially small joints. When the crystals deposited in joints, the body's defense mechanism; white blood cells attack on the crystals, causing inflammation and pain. Only 20% of hyperuricemia patients develop Gout (*Niqras*), Gout (*Niqras*) can affect any joint in the body, but flares often appear in MTP joints especially first MTP joint.

Physiological daily amount of endogenous and exogenous uric acid is about 700 mg, which is balanced by an equal output via feces and urine. 30% of uric acid is broken down by intestinal flora and expelled through the stool, while the remaining 70% (approximately 500 mg per day) is excreted unchanged through the kidneys. In most mammals, the enzyme uricase (urate oxidase) oxidizes uric acid to allantoin. Allantoin is highly soluble in water, therefore it does not accumulate in crystals and it is excreted unchanged through urine. Consequently, this makes urate oxidase very effective in lowering uric acid levels. Unfortunately, urate oxidase is not a functional human enzyme, probably due to mutations occurring during the myocene, and uric acid water-solubility is limited. As a result, humans but not other mammals can develop hyperuricemia and uric acid crystals can accumulate in human tissues and in the urinary tract, causing chronic hyperuricemia-related disease.

The patient having longstanding hyperuricemia generally develop into acute gouty arthritis, which is usually monoarticular. About 20% of the total cases of hyperuricemia converted in gouty arthritis, and the most common joint affected in first MTP joint (75%) and other small joints. The incidence of hyperuricemia tending to gouty arthritis is more in male than female.

Causes of High Uric Acide Levels

Primary Hyperuricemia: Increased production of uric acid from purine. When kidneys cannot get rid of the uric acid from the blood, resulting in high levels

Secondary Hyperuricemia: Certain cancers or chemotherapeutic agents may cause an increased turnover rate of cell death. This is usually due to chemotherapy, but high uric acid levels can occur before chemotherapy is administered. After chemotherapy, there is often a rapid amount of cellular destruction, and tumor lysis syndrome may occur. One may be at a risk for tumor lysis syndrome if he or she receives chemotherapy for certain types of leukemia, lymphoma, or multiple myeloma, if there is a large amount of disease present.

Kidney disease: Some time the kidneys also not been able to clear the uric acid out of system, thus causing hyperuricemia.

Medications: There are certain medicines that can cause increased levels of uric acid in the blood

Endocrine or metabolic conditions: Certain forms of diabetes or acidosis can cause hyperuricemia.

Elevated uric acid levels may produce kidney problems; or none at all. People may live many years with elevated uric acid levels, and they do not develop gout or gouty arthritis (arthritis means "joint inflammation"). Only about 20% of people with elevated uric acid levels ever develop gout and the most commonly affecting first metatarsophalangeal joint (MTP). Some people with gout do not have significantly elevated uric acid levels in their blood.

Foods that are rich in purine include

- All organ meats (such as liver), meat extracts and gravy.
- Yeasts, and yeast extracts (such as beer, and alcoholic beverages)
- Asparagus, spinach, beans, peas, lentils, oatmeal, cauliflower and mushrooms

Foods that are low in purine include

- Breads, pasta, flour, tapioca, cakes
- Milk and milk products, eggs
- Lettuce, tomatoes, green vegetables
- Cream soups without meat stock
- Water, fruit juice, carbonated drinks
- Peanut butter, fruits and nuts

Allopathic Medicines for Hyperuricemia

Uricosuric Drugs: These drugs work by blocking the reabsorption of urate, which can prevent uric acid crystals from being deposited in to body tissues. Examples of uricosuric drugs include probenecid and sulfinpyrazone.

Xanthine oxidase inhibitors: Xanthine oxidase inhibitors are substances that inhibits the activity of xanthine oxidase, an enzyme involved in purine metabolism. Xanthine oxidase inhibitors are of two types; purine analogues and others. Purine analogues include allopurinol, oxipurinol, and tisopurine. Others include febuxostat, topiroxostat, and inositols.

Symptoms of gout (*Nikras*) may include

- Severe joint pain
- Joint stiffness
- Difficulty moving affected joints
- Redness and swelling

If the condition of hyperuricemia stays for several months, uric acid crystals can form clumps called tophi. These hard lumps are found under the skin, around the joints, and at the top of ear. Tophi can worsen joint pain and over time damage joints or compress nerves. They're often visible to the eye and can become disfiguring.

Uric acid crystals can cause a buildup of stones in kidneys. Often, the stones are small and passed through the urine. Sometimes, they can become too large to pass and block parts of urinary tract.

Materials and Methods

The study was conducted at Kidwai Clinic, Lucknow, Uttar Pradesh. In view of the incidences, the cases having hyperuricemia along with gouty arthritis (*Niqras*) involving first MTP joint were only selected for the study. To evaluate the efficacy of Unani Complex Medicines (*Murakkab*) i.e. Habb-e-Asgand, Habb-e-Suranjan, and Sharbat Bazoori Motadil with and without Hijama-Bil-Shart, 30 cases of hyperurecemia with gouty arthritis of first MTP joint between the age group of 40 to 60 years of age in ratio of 70% male and 30% female were taken. These cases were divided in to three groups; Group A, Group B, and Group C (10 cases in each group). Unani Complex Medicines (*Murakkab*) including Habb-e-Asgand, Habb-e-Suranjan,

and Sharbat Bazoori Motadil were obtained from Herbo Pharmaceuticals, Allahabad along with Hijama Bil Shart. However a comprehensive study on types of cupping along with Unani Complex Medicines (*Murakkabat Advia*) needs to be carried out in the near future. The cases were included in the study according to Standard Unani Diagnostic Parameters *Alamaat-ul-Amzija* like built, weight, complexion, touch, hair colour, hair texture, diet, sleep, weather, pulse, temperature, blood pressure, respiratory rate, emotional behavior along with modern diagnostic parameters were used based on the external manifestations including temperament of individuals.

The diagnostic parameters used for inclusion of the patient in the study included serum uric acid, serum urea and creatinine, sugar fasting and PP, LFT, CBC, ESR, C-reactive protein, ASO titer, RA factor along with x-ray of the affected joint apart from the clinical signs and symptoms. The clinical signs and symptoms were recorded on every 15-day visit along with ESR and serum uric acid level. Serum uric acid, ESR, serum urea, creatinine, C-reactive protein along with hepatic profile were done before and after the study to evaluate safety of the trail.

Patients having liver diseases like hepatitis, jaundice, and cirrhosis, heart disease like ischemic heart disease, renal diseases like acute or chronic kidney disease, nephritis, renal failure, anemia, diabetes mellitus, pregnancy, and lactating women were excluded from the study.

Of 30 patients of the study, the cases were divided in three groups i.e. 10 cases (33.34%) in each group. First group of 10 patients (33.34%) included the trail of only Unani Murakkab Advia, second Group of 10 patients (33.34%) included Unani Murakkab Advia with Hijama-Bil-Shart, and the third group of 10 patients (33.34%) were kept on placebo. The percentage of male female ratio were 70% male and 30% female as it is less likely in female than male. However, the sample size was small; the negative demographic observations need to be evaluated for any change in the demographic presentation of disease by further studies with a large sample size.

Patients were randomly placed in three groups. Group A was administered Habb-e-Asgandh Two tablets three times a day, Habb-e-Suranjan two tablets three times a day, and Sharbat Bazoori Motadil 20 mL thrice a day with normal water. Group B was administered Habb-e-Asgandh Two tablets three times a day, Habb-e-Suranjan two tablets three times a day, and Sharbat Bazoori Motadil 20 mL thrice a day with normal water along with Hijama Bil Shart on the affected joint (First MTP joint) at Zahrul qadam (Point No. 129) with placement of cup No. 2 (Outer diameter 3.2 cm). Group C was placed on placebo, maintaining confidentially at both ends. Prior written consent was obtained on prescribed format. Weekly assessment was carried out according to the questionnaire depicting all parameters. Total duration of therapy was for three months. After completion of three months therapy, relevant serological tests were done and compared with findings recorded earlier. The recorded data were statistically analyzed by using period test.

Table 1: (Group A) Serum Uric Acid level pre-treatment and post-treatment

No. of Patients	Serum Uric Acid level (Pre-treatment)	Serum Uric Acid level (Post-treatment)		
(n=10)		(Ist Month)	(2nd Month)	(3rd Month)
1.	6.7 g/dL	6 g/dL	5.5 g/dL	5.0 g/dL
2.	7.2 g/dL	6.9 g/dL	6.1 g/dL	5.7 g/dL
3.	5.8 g/dL	5.5 g/dL	5.1 g/dL	4.3 g/dL
4.	6.2 g/dL	5.9 g/dL	5.5 g/dL	5.1 g/dL
5.	7.9 g/dL	7 g/dL	6.8 g/dL	6.0 g/dL
6.	6.6 g/dL	6.5 g/dL	6.1 g/dL	5.4 g/dL
7.	6.2 g/dL	5.6 g/dL	5.1 g/dL	4.7 g/dL
8.	7.1 g/dL	6.6 g/dL	5.8 g/dL	5.1 g/dL
9.	5.7 g/dL	5.2 g/dL	4.8 g/dL	4.5 g/dL
10.	7.7 g/dL	7.0 g/dL	6.8 g/dL	6.8 g/dL

Table 2: (Group B) Serum Uric Acid level pre-treatment and post-treatment

No. of Patients	Serum Uric Acid level (Pre-treatment)	Serum Uric Acid level (Post-treatment		
(n=10)		(Ist Month)	(2nd Month)	(3rd Month)
1.	6.1 g/dL	6 g/dL	5.5 g/dL	5.0 g/dL
2.	6.8 g/dL	6.9 g/dL	6.1 g/dL	5.7 g/dL
3.	5.8 g/dL	5.5 g/dL	5.1 g/dL	4.3 g/dL
4.	6.1 g/dL	5.9 g/dL	5.5 g/dL	5.1 g/dL
5.	6.9 g/dL	6.1 g/dL	5.8 g/dL	5.5 g/dL
6.	8.2 g/dL	7.5 g/dL	7.0 g/dL	6.3 g/dL
7.	6.4 g/dL	6.3 g/dL	5.5 g/dL	4.9 g/dL
8.	6.9 g/dL	6.6 g/dL	5.8 g/dL	5.4 g/dL
9.	7.3 g/dL	6.8 g/dL	6.1 g/dL	5.4 g/dL
10.	7.3 g/dL	7.0 g/dL	6.3 g/dL	5.7 g/dL

Table 3: (Group C) Serum Uric Acid level pre-treatment and post-treatment

No. of Patients	Serum Uric Acid level (Pre-treatment)	Serum Uric Acid level (Post-treatment)		
(n=10)		(Ist Month)	(2nd Month)	(3rd Month)
1.	6.1 g/dL	6.1 g/dL	6.3 g/dL	6.9 g/dL
2.	6.8 g/dL	6.9 g/dL	6.7 g/dL	6.7 g/dL
3.	5.5 g/dL	5.8 g/dL	5.7 g/dL	6.0 g/dL
4.	5.6 g/dL	5.4 g/dL	5.6 g/dL	5.7 g/dL

5.	7.9 g/dL	8.1 g/dL	8.4 g/dL	8.9 g/dL
6.	10.8 g/dL	10.7 g/dL	10.3 g/dL	10.4 g/dL
7.	5.9 g/dL	5.9 g/dL	5.9 g/dL	5.7 g/dL
8.	5.3 g/dL	5.6 g/dL	6.2 g/dL	6.2 g/dL
9.	7.7 g/dL	7.8 g/dL	7.4 g/dL	7.9 g/dL
10.	6.4 g/dL	6.7 g/dL	6.7 g/dL	7.0 g/dL

Resolution of Sign/Symptoms with Unani Murrakab Advia without Hijama (Group – A) (n=10) (Pain Score Scale 1-10)

(i) Post treatment in First Month of therapy				
No of patients	Pain	Redness	Stiffness	Immobility
10	30%	25%	25%	10%
(ii) Post treatment in Second Month of Therapy				
No of patients	Pain	Redness	Stiffness	Immobility
10	50%	40%	50%	30%
(ii) Post treatment in Third Month of Therapy				
No of patients	Pain	Redness	Stiffness	Immobility
10	70%	75%	70%	70%

Resolution of Sign/Symptoms with Unani Murrakab Advia with Hijama Bil Shart (Group – B) (n=10) (Pain Score Scale 1-10)

(i) Post treatment in First Month of therapy				
No of patients	Pain	Redness	Stiffness	Immobility
10	80%	60%	50%	50%
(ii) Post treatment in Second Month of Therapy				
No of patients	Pain	Redness	Stiffness	Immobility
10	90%	80%	80%	70%
(ii) Post treatment in Third Month of Therapy				
No of patients	Pain	Redness	Stiffness	Immobility
10	100%	100%	90%	95%

Results

Patients in Group A showed a positive response in declining of serum uric acid levels with less improvement in overall inflammation of the affected joint. In Group B there is also positive response in declining of serum uric acid levels but there is dramatic improvement in signs and symptoms of pain, swelling, redness, and stiffness of the joint. There is marked reduction of overall inflammation of the joint and increase in movement of the joints. Group C shows no response to overall therapy, there is comparatively marked progression of disease.

Discussion

In this study, the efficacy of Unani Murakkab Drugs with and without Hijama bil Shart was evaluated for over a period of three months. The above observation shows that the Unani Murrakab Advia not only reduces inflammation of the joint but also lowering uric acid level by high excretion of serum uric acid through the urine. The study also revealed that the drug has no effect on vital signs, i.e. heart rate, blood pressure, body temperature, respiratory rate, and weight of the patient. During the study very minimal incidence (2%) patients complaining of gastric upset leading to hyperacidity, flatulence, abdominal cramping, and diarrhea. On the basis of improvement in the clinical subjective parameters including uric acid level, pain, swelling, inflammation, redness, restriction of movement. In patients of gouty arthritis; the inflammation is due to the collection of morbid material (*ghair tabai madda*) around the affected joint. Use of Hijama on the affected joint causing evacuation and dispersion of morbid material and improves the blood circulation to the affected area leading to decrease in overall inflammation.

Conclusion

The study clearly reveals the efficacy of Unani Murrakab Advi with Hijama-bil-Shart for reducing serum uric acid level along with gouty arthritis of first metatarsophalangeal (MTP) joint. The efficacy of any treatment depends upon how well the patient tolerates it and how long he can undergo treatment without experiencing any serious side effects. The present study creates ample room for further studies.

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