Journal of Arthritis
ISSN: 2167-7921

Akhtar et al., J Arthritis 2017, 6:6
DOI: 10.4172/2167-7921.1000256

Review Article Open Access

Role of Hijama' *(Cupping Therapy) in* the Management of Niqras (Gouty Arthritis)

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Received date: October 18, 2017; **Accepted date:** November 01, 2017; **Published date:** November 04, 2017

Abstract

Gout is a potentially progressive and debilitating form of chronic inflammatory arthritis caused by the deposition of monosodium urate crystals in synovial fluid and other tissues, characterized by sudden & severe episode of pain, warmth and swelling in a joint. People suffering from recurrent attacks frequently experience pain and disability, reduced health-related quality of life and productivity and increased morbidity. NSAIDs, colchicines, glucocorticoids, uric acid lowering agent like uricostatic drugs (allopurinol, a xanthine oxidase inhibitor), Uricosuric drugs (Probenecid) are very effective but risky in patients having pre-existing renal, cardiovascular, gastrointestinal and metabolic disorders. Cupping (Al-Hijama) is a widely used therapeutic regimen of Unani system of medicine with high acceptance in Egypt and Arab countries. It is used for the treatment of various inflammatory and painful conditions like sciatica, gout, rheumatoid arthritis, pain of knee, removal of deep swelling, disease of liver and skin etc. It is a minor surgical excretory procedure related scientifically to the principles of renal glomerular filtration and abscess evacuation where a pressure-dependent excretion of causative pathological substances occurs. Cupping is thought to act mainly by increasing local blood circulation and relieving the painful muscle tension. It acts by improving microcirculation, promoting capillary endothelial cell repair, accelerating granulation, and angiogenesis in the regional tissues. It is the best deep tissue massage which normalizes the patient's functional state and progressive muscle relaxation. In Unani system of Medicine, this is a simple and economic treatment, effectively treating diseases with different etiologies and pathogenesis.

Keywords: Gout; Cupping; Unani system of Medicine; NSAIDs

Introduction

Gout (Niqras) is one of the oldest known and most common forms of arthritis. Hippocrates (Buqrat, 460-377B.C), the father of medicine, called it the "Disease of King 'for its association with wealthy men who overindulged in food and drink [1-3]. Gout was also known as the "King of Diseases" because of its unbearable pain [2,3]. The term gout is derived from the Latin word-gutta, meaning drop, based on the belief that gout is caused by a poison falling drop by drop into the joint [2,4,5]. Gout, as we understand it today, is a clinical syndrome characterized by an inflammatory response to monosodium urate crystals [2].

According to Ibn Hubl (1121-1213 AD), Niqras is derived from the term 'Naqoroos' which means 'the joint of great toe', from which it derives its name [6-9].

Gouty arthritis, one of the most primitive diseases recognized as a clinical entity, was first documented by the Egyptians in 2640 BC. Hippocrates referred to gout as "the unwalkable disease". He also highlights the relation between the disease and life style, referring gout to be "arthritis of rich" [10,11].

During the Arab era of Unani Medicine, gout was described by most of the scholars but none of them added much to the work of Hippocrates and Galen (Jalinus, 131-200AD). After that Al-Tabbari (Rbban Tabri, 780-850 A.D) in Paradise of Wisdom (Firdaus- Al-Hikmat) and Rhazes (Zakariya Rhazi, 850-923 A.D) in Liber Continens (Al-Hawi-Fi-Tibb) added some precious details to this disease [12,13].

In practice, hyperuricemia is defined as a serum urate level in excess of 7 mg/dl in adult male and 6.0 mg/dl in adult female. Gout constitutes only one-tenth of patients of hyperuricemia [2,14,15]. It may result from the over production (10%) or the under excretion (90%) of urate and often from a combination of the two [16].

Gout is a disorder of purine metabolism, mainly affecting middle-aged to elderly men and postmenopausal women. It is associated with the presence of Monosodium Urate (MSU) crystals in synovial fluid or tissue, particularly the metatarsophalangeal joint of the first toe is often involved, but tarsal joints, ankles, hands and knees are also commonly affected [17-21].

Nowadays, owing to the application of modern technology in medicine, the deposition of crystals of uric acid is known to be the main factor in pathophysiology of patients [22]. The drugs that have proven to be effective against acute gout are Non-steroidal anti-inflammatory drugs or colchicines. The common options to prevent gout are the drugs like Allopurinol or sulphinpyrazone. But all of the above stated drugs exhibit serious systemic side effects [23].

In Unani system of Medicine, cupping therapy is a simple and economic treatment which effectively treated diseases with different etiologies and pathogenesis. Cupping therapy is popular as '*Al-Hijama*' in Egypt and Arab countries. It is beneficial in various inflammatory joint disorders including gout and other forms of arthritis.

Epidemiology

Hippocrates was the first to identify the hereditary tendency of gout. His observation has been well proven even nowadays [12,24]. Hereditary tendency occurs in 50-75% of cases [25].Gout is more frequently inherited from the father's side than that of the mother [26].

· Hyperuricemia
· Age
· Central Obesity
· High Protein diets
· Alcohol (Beer)
· Medicine: Diuretics, Pyrazinamide, Ethambutol, Aspirin in high dose etc.
· Some local factors: ↓Temperature, ↓Ph etc.

Table 1: Common risk factor.

The single most important factor suggestive of the potential risk of development of gout is the serum uric acid concentration. There are various other factors (as shows in Table 1) that contribute to the higher serum urate levels, such as alcohol intake, dietary excesses, seafood, high intake of meat, surgical trauma, sepsis, stress starvation, dehydration, body weight, age, blood pressure, drugs like diuretics, pyrazinamide and aspirin [14,27,28,29]. However, the epidemiology of gout varies in accordance to the alteration in lifestyle, drugs, climate conditions and cyclic changes and rising span of life [28]. Dietary habits like increased intake of sugar sweetened soft drinks and fructose may also correspond to the development of gout [1].

Conditions Associated with Gout

Conditions which are commonly associated with gout are obesity, metabolic disorders, hypertension, nephrolithiasis, renal failure, urate nephropathy, cardiovascular disease, type IV hyperlipoproteinaemia and type 2 diabetes [2,3,14,15,27,29]. Gout was linked with a 60% increased risk for coronary artery disease (CAD) in men in the Framingham Heart Study and 26% increased independent risk of myocardial infarction. Men with gout have a 41% increased risk for incident type 2 diabetes. Men with gout also have an increased risk of death from all causes [2]. There appears to be a considerable increased occurrence of hypothyroidism and cerebrovascular diseases among the patients of gouty arthritis [16].

Incidence of Gout

Gout is one of the most common types of inflammatory joint disease; affecting an estimated 1-1.5 % of the world's population [30]. Gout affects more than 1% of adults in the western countries. Male: female ratio ranges from 7:1 to 9:1. In younger age groups (less than 65 years), the prevalence is four times higher in men than women (4:1), but in older age groups (more than 65 years), the gap tapered e.g. one woman for every three men with gout (3:1). The Bhigwan COPCORD survey demonstrated low prevalence of gout (0.12%) in rural India. In another Indian study, prevalence of 2% was recorded [16]. The data of US Veterans Administration Normative Ageing Study indicated that the incidence of gout rose quickly from 0.9/1000/year in men with a serum urate level less than 7 mg/dl to 4.1/1000/year in those with serum urate level 7 to 8 mg/dl and 49/1000/year with serum urate level more than 9 mg/dl[2].

Prevalence

Currie et al. were the earliest to report on the prevalence of gout and established that from 1971 to1975, 0.26% of the population of Great Britain had gouty arthritis. A higher prevalence (0.95%) of gout in England in 1990s was reported by Harris et al. recently, data collected from the United Kingdom (UK) General Practice Research database indicated that the prevalence of gout in England was 1.4% from 1990 to 2005. Kuo et al., in 2012 monitored a higher prevalence of gout e.g. 2.5% in their analysis of medical records and described that the prevalence of gout had risen by 63.9% since 1997. According to a study done in Taiwan, prevalence of self-reported gout raised from 4.7% to 8.2% in men over the periods 1993 to 1996 & 2005 to 2008. In a study in Southern Taiwan, Physician-diagnosed gout was viewed in 2.9% of the Hanese population in Taiwan. Related surveillances have been made about the higher prevalence of gout in the United States, New Zealand and Australia [31].

The prevalence of gout is found to have increased in recent years. It is rare in children and premenopausal women [14]. The epidemiological data showed that the incidence and prevalence of gout had more than doubled in last forty years. According to UK General Practice, the prevalence of gout is roughly 1.4% while serum urate levels more than 7 mg/dl are found in up to 18% of the population [2]. The incidence of gout varies in the population with an overall prevalence of less than 1 to 15.3%. The prevalence increases substantially with age and increasing serum urate levels. For serum urate values greater than 9 mg/dl, the cumulative incidence of gout reaches 22% after 5 years [3,16].

Hippocrates mentioned that-gout is very common in men but may be seen in women after menopause [1]. Eunuchs do not take the gout, not become bald, A teenager does not have gout unless he indulges in coitus. Gouty inflammation subsides within 40 days [3,6,12,32,33].

Classification of Gout

Gout is often classified as primary or secondary. Primary gout accounts for 95% of the total cases and is either due to primary overproduction (10%) or under excretion (90%) of uric acid. Secondary gout (5%) results from a demonstrable medical disorder, leading either to overproduction or defective excretion of uric acid (Figure 1) [1,14,15].

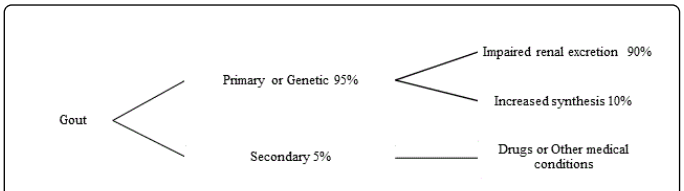

Figure 1: Classification of gout.

Pathophysiology

Most of the Unani Physicians discussed gout along with various other arthropathies like Arthralgia (Wajaul Mafasil) and Sciatica (Irq-un-Nisa).

Galen (129-200AD) stated that arthralgia, sciatica and gout belong to the same group and their name signifies the different areas of affliction [34].

Avicenna (980-1037 AD, Ibn-e-Sina) in his famous book Alcanon (Alqanoon fit Tibb) explains that, the Humours (Akhlat) liable for Niqras may be blood (Dam) only, or blood mixed with phlegm (Dam-e-balghami), or blood mixed with yellow bile (Dam-e-safrawi), or blood mixed with black bile (Dam-e-saudawi) or a mixture of humours [35]. Rhazes blames raw phlegm (kham balgham) as a causative humour of Niqras [12]. Nuh Al Qamri, author of Ghina Muna says that Niqras results from the dissemination of humours towards the extremities, which is repulsed by the vital organs and accepted by extremities [34].

Uric acid is derived from the purine degradation. There are two main sourcesof purines e.g. endogenous (de novo synthesis) and exogenous (dietary purines).The body uric acid concentration depends on a balance between synthesis and ingestion and excretion of uric acid (Figure 2). 75% of the uric acid is excreted in the kidneys and the remaining is lost in the gut. The uric acid is freely filtered at glomerulus and 90% is then reabsorbed. Renal handling of uric acid involves four step process- glomerular filtration, proximal tubular reabsorption (PCT), tubular secretion, and postsecretory reabsorption (Table 2) [2,27,29]. In hyperuricemia; urate is usually present in the blood as a supersaturated solution. Lowering of the pH lessens the solubility and increases the likelihood of crystal formation. There are various factors that influence the solubility of urate as showed in Table 3. [2,27,36].

Conditions that promote PCT Reabsorption of Uric acid
· ↓ uric acid clearance
· ↑ serum uric acid level
Conditions that decrease PCT Reabsorption of Uric acid
· ↑uric acid clearance
· ↓ serum uric acid level

Table 2: Factors affecting the renal reabsorption.

Temperature
pH
Cations concentration
Level of articular dehydration
Presence of nucleating agents e.g. None aggregated proteoglycans, insoluble collagens, chondroitin sulfate.

Table 3: Factors influencing the solubility of urate in joints 20.

Urinary urate excretion more than 1000 mg/day indicates over production which can potentially result from several inborn errors of metabolism, malignant diseases, especially when treated with anticancer drugs, chronic and acute leukemia, myeloma, polycythemia, and others. Under excretion in which urinary urate excretion is less than 330 mg/day can result from decreased urate clearance due to reduced urate filtration, enhanced reabsorption or decreased secretion [2,3,14,15,27,29].

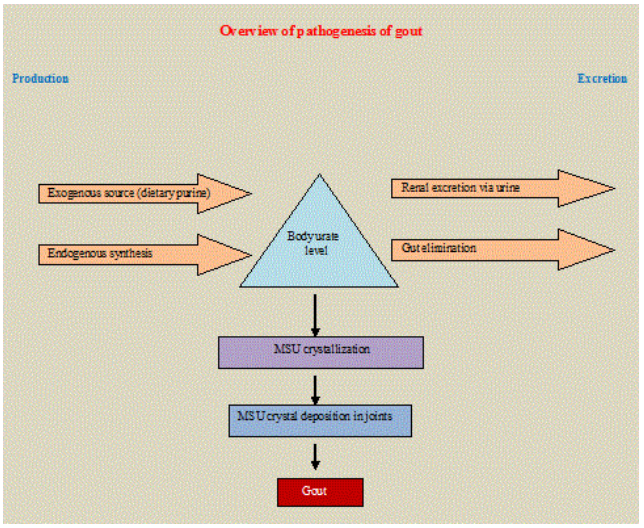

Figure 2: Overview of pathogenesis of gout.

Uric acid is a weak acid present as urate (ionized form) in physiological fluids [14]. At concentrations more than 6.4 mg/dL, urate can crystallize as MSU in and around joint and tissues. Gouty arthritis is aggravated/ caused by the activation of numerous inflammatory mediators by MSU crystal. MSU crystals are released into the joint cavity and are phagocytosed by synovial lining cells and infiltrating neutrophils. These cells release proinflammatory cytokines such as TNF-@ (Tumor necrosis factor), IL-1, IL-6, IL-8 which promote recruitment of additional neutrophils to the site of crystal deposition, thereby inducing an intense inflammatory response [1,14,15,16,37,38].

Neutrophils are attracted to synovial fluid by crystal induced chemotactic factors C5a, leukotriene B4 and IL-1. Polypeptide like C1q, C1r, C1s fibronectin, fibrinogen, IgG, Lysosomal enzymes, apolipoprotein and TGF-b which have been identified to mediate inflammatory response. Other mediators released from polymorphonuclear leucocytes which appear to have an important role in the inflammatory response include LTB4, kinins, collagenase, kallikrein, prostaglandin E2, and IL-116.The joint inflammation is self-limited, although auto-limitation of active inflammation is brought out by macrophages, neutrophil necrosis, and apoptosis [14] in Figure 3.

Clinical Phases of Gout

Gout is classified into four stages:

1) Asymptomatic hyperuricemia

2) Acute gouty arthritis

3) Intercritical gout

4) Chronic tophaceous gout

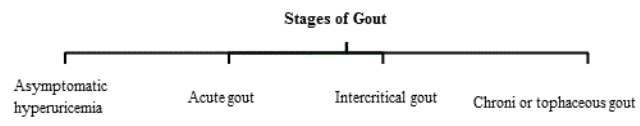

Figure 3: Stages of gout.

The basic pattern of clinical gout begins with acute attacks of intensely painful arthritis. The first attack is usually monoarticular and is associated with few constitutional symptoms [1,2,3,14,16].

Asymptomatic hyperuricemia

It is characterized by high serum uric acid level but gout as manifested by arthritis symptoms, tophi, or uric acid nephrolithiasis has not yet appeared. Most patients remain asymptomatic throughout their lifetime [1,16].

Acute gouty arthritis

The first episode of acute gouty arthritis develops between fourth and sixth decades in men and after 60 years in female. Acute gouty arthritis usually presents as a rapid onset of severe monoarthritis, repeatedly at night. In ninety percent cases, the first attacks are monoarticular and first metatarsophalangeal (MTP) joint is typically affected (podagra). Other joints may be involved including the ankle, knee, wrist, and elbow as well as the small joints of the hands and feet. The joints and surrounding tissues are swollen, hot, red, shiny, tender and enormously painful. There is mild fever with chills [1,2,14,16,39,40]. Polyarticular involvement may also occur and is more common in joints with preexisting injury from trauma or another type of arthritis such as rheumatoid arthritis [1] in Table 4.

First metatarsophalangeal joint (MTP) 90%
Ankles
Heels
Knees
Wrist
Fingers
Elbows

Table 4: Joints commonly affected by gout.

Intercritical gout

The term has been applied to the period between gouty attack and this may last for months or even years. During this phase, MSU crystals have been demonstrated in the synovial fluid [1,2,16] Table 5 & Figure 4.

Alcohol intake
Ingestion of high purine foods
Surgery
Joint trauma
Any acute illness
Sepsis
Starvation
Haemorrhage
Odd exercise

Table 5: Factors precipitate an acute attack of gout [2,16].

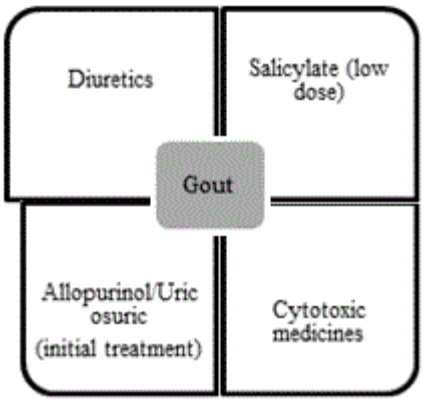

Figure 4: Medicines that precipitate an acute attack of gout.

Chronic tophaceous gout

Eventually, the patient may enter a phase of chronic polyarticular gout with no pain free intercritical periods [16,41]. The rate of formation of urate crystals (tophi) is related to the length and severity of hyperuricemia and accompanying renal disease [42]. Tophi may form anywhere but are found most commonly in the finger pads, wrist, pinnae, knee, olecranon bursae and over pressure points [1,2,16, 43].

Complications

Renal complications

Renal damage is the most common extra skeletal complication of gout. Kidneys are the only vital structures probable to be affected and renal failure (commonest cause) accounts for 10 to 25% of deaths of gouty patients. Renal damage is the most frequent complication of hyperuricaemia. Chronic urate nephropathy is due to the deposition of MSU crystals in the renal parenchyma and acute nephropathy is due to the obstruction of renal tubules by urate crystals or uric acid calculi [14,15].

Cardiovascular complications

Gout tends to shorten life mainly because of the cardiovascular changes and the resulting kidney ailment with hypertension. Arteriosclerotic changes in the coronary arteries and in the cardiac valve are common [25]. Patients, who have received a kidney or heart transplant and are receiving anti-rejection therapy with cyclosporine

A, often along with diuretics, are at an increased risk of developing gout [2].

Skin complications

Gouty patients are particularly liable to develop eczema, boils and other skin infections due to high incidence of uric acid in blood, which increases the acidity of blood and perspiration [25,26]. Cutaneous tophi, with or without inflammation, can develop in elderly women receiving diuretics. Urate crystal deposits may be associated with an unusual panniculitis [2].

Respiratory complications

Pulmonary disorders such as asthma and chronic bronchitis, pneumonia [25,26].

Gastrointestinal complications

Such as dyspepsia, loss of appetite, constipation, unpleasant taste in the mouth, flatulent colic [25,26].

Spinal complications

Tophi of various sizes can develop in any anatomic structure of spine leading to nerve root compression, cord compression or even lumbar spinal stenosis. Tophi sometimes lead to peripheral nerve root compression, for example, carpal tunnel syndrome or cubital compression. The diagnosis can be confirmed by imaging with a CT scan, MRI or ultrasound [1,2].

Miscellaneous

Catarrh of nose and throat, phlebitis are also frequent among the gouty individuals [25,26].

Diagnosis

The patient's family history, clinical symptoms, presence of tophi, and radiological findings should be recorded to confirm the diagnosis. The strengthened diagnosis is made on the basis of following parameters.

Synovial fluid examination

A cell count, Gram stain, culture, and crystal analysis should be carried out on synovial fluid to prove the diagnosis [1,2,15,16,27].

Serum uric acid level

A high level with characteristic history is almost diagnostic of gout. However, serum uric acid level alone does not justify the diagnosis of gout, because there are many other factors which elevate uric acid level and it may be normal during an acute attack [1,2,15,16,27].

Polarised light microscopy

It is a gold standard to diagnose the acute gout. Diagnosis can only be made with certainty by demonstrating strongly negative birefringent needle & rod shaped MSU crystals in joint fluids when viewed under the compensated polarised light microscope [1,2].

Measurement of urine, uric acid in 24-hour

Patients who excrete more than 800 mg of uric acid in 24-hours on a regular diet are over excretors and over producers of uric acid and the patients excreting more than 1,100 mg uric acid in 24-hours demand the monitoring of their renal function because of the risk of calculi and urate induced nephropathy [16].

Blood

Lipid profile, liver function tests, blood sugar, renal function tests to screen comorbidities and cardiovascular risk factors [1].

Urine analysis

Gouty and hyperuricaemic patients have more incidence of urinary caculi [1,2].

Radiography

It gives little information to diagnose the acute gout but can be sometimes helpful in ruling out alternative diagnosis. Radiographs are usually normal or show nonspecific changes around the joint in early stages of gout. In chronic gout, radiograph shows characteristic changes such as punched out erosions with overhanging edges mostly para-articular (Table 6) [2,14,27,44], asymmetrical and eccentric [1,2,14].

S. N	Disease	Description
1	Septic arthritis	· Etiology-Pyogenous organism (Staphylococcus aureous)
		· Gender - Commonly in male
		· Onset- Acute with features of septicemia
		· Site- Knee (most commonly) others hip, shoulder, elbow etc.
		· Presentation-Severe throbbing pain, a single large joint (monoarticular) commonly involved or multiple small joints.
		· Investigations-Blood shows neutrophilic leucocytosis, ↑ ESR, Culture confirmed causative organism, Joint aspiration shows features of acute septic Inflammation like WBC>10000, PMN leucocytosis >75%.
		· Etiology-Unknown or Genetic
		· Gender-Female are threefold more prone than male patients
		· Age- 20 to 50 years
		· Onset-Insidious
2	Rheumatoid arthritis	· Site-It starts from smaller joints of hand and foot like metacarpophalangeal (MP) joint of hands, proximal interphalangeal (PIP) joints and may spread to knee, ankle, elbow, wrist etc.
		· Presentation-Migratory polyarticular, bilaterally symmetrical, joints stiffness (morning).
		· Investigations-Decalcification & reduced joint space in x-ray, ESR increase, positive RA factor.

		· Etiology- Multifactorial e.g. mechanical, genetic, metabolic
		· Gender - Male
		· Age- over 40 years
3	Osteoarthritis	· Onset-Insidious
		Site- Weight-bearing joint like knee, hip commonly, distal interphalangeal (DIP) joints of finger are often involved.
		· Presentation-Asymmetrical affection of one or more joints. Pain is the earliest symptom, intermittent in beginning & become constant later.
		· Investigations-Diminished joint space with osteophytes.
		· Presentation- like rheumatoid-polyarthritis, distal IP joints of hand affected.
4	Psoriatic arthritis	· Classical skin lesions help in diagnosis.
5	Traumatic arthritis	· H/O trauma positive, acute onset
6	Pseudo gout	· Presentation- like gout.
		· Sodium pyrophosphate crystal deposition.
7	Xanthomatosis	· The clinical presentation, presence of hypercholesterolemia, and absence of MSU crystal within the joints will differentiate this condition from gout

Table 6: Differential diagnosis.

Management

The management of gout includes both nonpharmacological and pharmacological modalities. Several approaches are existing to treat gout depending upon appearance of the disease, its clinical phase, specific risk factors, and general risk factors such as age, sex, obesity, alcohol consumption, drug interaction. Rapid relief of pain and inflammation and reducing serum urate level is the crucial aim of treatment in case of gout [2].

Numerous drugs used for gout include: NSAIDs, colchicines, glucocorticoids, uric acid lowering agent like Uricostatic drugs (Allopurinol, a xanthine oxidase inhibitor), Uricosuric drugs (Probenecid). All these drugs are very effective but are risky in patients who have pre-existing renal, cardiovascular, gastrointestinal and metabolic disorders. Sometimes, the undesirable effect of these drugs, especially in old patients who have the highest incidence of gout can be dangerous and may produce life-threatening hypersensitivity reactions [2,15].

Prolonged steroid therapy causes osteoporosis, hypertension, steroid diabetes, gastric ulcer, purple striae, muscular weakness, susceptibility to infections, delayed healing, psychiatric disturbances and steroid dependence [45].The drawback of all above mentioned drugs call for the development of some other option for the management of gout with superior efficacy and lesser toxicity. Unani system of medicine or Greco-Arabian medicine is a traditional alternative system of medicine which has played an important role in human health and welfare. Unani medicine is based on the Hippocratic doctrine of four humours (Akhlat) i.e., Dam (blood), Bulgham (phlegm), Safra (bile), Sauda (black bile). A proper balance of Akhlat within the body avoids the upsurge of toxins, and sustains best health. Humoural imbalance is often a root basis in the origin and progress of a specific disease. The key principles of management in Unani system of medicine are Ilaj-bit-Tadbeer wa Taghziya (Regimental and Diet therapy), Ilaj-bid-Dawa (Pharmacotherapy), and Ilaj-bil-Yad (Surgery) [46]. Most of the Unani Physicians advised Ilaj-bit-Tadbeer or regimental therapy to treat the gouty pain which includes Fasad (venesection), Cauterization (Kai), Leeching (Irsal-e-Alaq), Nutool, Hammam (Bathing), Dalak (Massage), Riyazat (Exercise), Qay (emesis), Ishal (purgation) etc. [47,48]. Cupping is one of the part of Ilaj-bit-Tadbeer and can relieve pain of gouty arthritis [49,50]. Hijamah is a very old method of treatment carried out by creating vacuum on suction which dates back to Hippocrates (460-377 B.C.) With the advent of technology, Hijamah is also proven to be valuable scientifically in a lot of diseased conditions [46].

Hijamah (cupping therapy) refers to Unani regimental approach of treatment. The literal meaning of Hijamah is to suck and can be largely classified into Hijamah-bish-Shart (wet cupping/ cupping with scarification) and Hijamah-bila-Shart (Dry cupping/ cupping without scarification).

The description of Hijamah is also found in the famous Egyptian Ebers Papyrus (1550 B.C). Hippocrates used cupping based treatment for various musculoskeletal disorders50.The Hijamah word gets derived from the word'Al-Hajm' which means to suck. It is a Unani regimental approach of treatment carried out by the use of specific type of vacuum cup or plastic vessels which adheres to the body surface, creating vacuum by heat or by unique suction equipment in order to remove the morbid materials from the body, to get back a displaced organ to its normal location, to redistribute the material from the diseased part or to promote the blood flow to the place of Hijamah [50,51]. In ancient times, horns of animals were used as a cup in cupping therapy. Hijamah should be done in the mid of lunar month because the humours are completely agitated at this time and is always done in the afternoon as it is most optimal time of the day [46].

Discussion

Cupping is a widely used therapeutic regimen of Unani system of medicine. It's used for the treatment of various inflammatory and painful conditions like sciatica, gout, rheumatoid arthritis, pain of knee, removal of deep swelling, disease of liver and skin etc. [46,48-50].

Wet cupping of Hijamah evacuates the morbid matter from affected area, advances blood circulation of affected part and gives superior nutrition to the area where cup is applied. Hence, swelling, tenderness etc. is alleviated. The dry cupping resolves swelling by redistribution of morbid humors from one site to other [48,50]. Wet cupping is just like a minor surgical excretory procedure scientifically related to the principle of renal glomerular filtration [49].

Hijamah (wet cupping) increases blood circulation, decreases congestion and prevents inflammatory extravasations from the tissues. Cupping is a type of excretory therapy and it eliminates blood and tissue fluid mixed with causative pathological substances (CPS) [49,50].

In wet cupping therapy, the excretion of causative pathological substances (CPS) occurs under pressure. CPS includes disease-causing substances that are produced during the disease pathogenesis. In this procedure, cups are placed over the skin and negative pressure is

applied which causes local gathering of filtered and interstitial fluid containing CPS at skin uplifting site within the cup. Cupping causes a pressure gradient and a traction force across the skin and capillaries to excrete collected fluid containing CPS. In this way, it causes clearance of blood and interstitial spaces from CPS. Wet cupping is involved in reactive hyperemia, opening in skin barrier, enhancing natural excretory function of skin, improving capillary and lymphatic circulations and restoration of homeostasis [49,50]. Wet cupping is also liable in production of nitric oxide [49]. It may also affect the autonomic nervous system and aid in reducing pain. The functional mechanism of wet cupping is related to the natural excretory function of kidney. Hence wet cupping is regarded as an artificial kidney that performs skin capillary filtration and emission of harmful particles and CPS at high pressure than that of the pressure present in renal glomeruli. In this way, wet cupping enhances the natural excretory function of skin [49]. Negative pressure suction and NO (released during cupping) may cause dilatation of local blood capillaries. This advanced microcirculation, increased permeability of capillaries, increased excess fluid drainage increases clearance and flow of lymph. Decreased capillary absorption at venous end, increase filtration of fluid at both arterial and venous end of capillary, increased excretion of fluid causes increased clearance of blood, plasma, lymph and interstitial spaces.

All of the above phenomenon leads to a decrease in the pressure of interstitial fluid, decrease in venous return and pressure, increase in speed of blood flow, decrease in congestion, improves circulation of blood and lymphatic circulation and resolution of swelling due to removal of CPS, prostaglandins, morbid matters (akhlat raddiya), and inflammatory mediators. All of the above effects may advance oxygen supply and perfusion of tissue, protect underlying structure, relieve muscle spasm and restore physiological balance of humours (akhlat), thereby providing relief from gouty pain and swelling [49].

Skin injury during cupping therapy leads to the release of β-endorphin (endogenous analgesic opioid) and adrenocortical hormones into circulation. Both adrenocortical hormones and β-endorphin could be supportive in blocking inflammation. Skin nerve endings are bathed in collected fluids which may cut their stimulation. On the basis of this, skin injury during cupping has an analgesic outcome by the release of endogenous opioids [49].

Conclusion

In addition to drug therapy, cupping therapy which is a part of Ilaj-bit-Tadbeer is a specific therapy in the traditional healthcare system. A favorable balance between various humours (Akhlat)/vital parameters after cupping is restored by a specific mechanism. Cupping helps to align the blood flow of skin and remove impure blood containing CPS from the diseased part, thereby proving to be beneficial in various inflammatory joint disorder including gout and other form of arthritis. It is also commonly used in relieving pain, muscle tension, and improve motion range. Cupping is the best deep tissue massage which normalizes the patient's functional state and progressive muscle relaxation.

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