

## A CRITICAL REVIEW ON VATARAKTA W.S.R TO HYPERURICEMIA

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## ABSTRACT

*Vatarakta* is an illness where both *Vata* and *Rakta* are afflicted by distinct etiological factors. Description of *Vatarakta* has been done since Samhita Kala. According to Aacharya Charaka, clinical features of *Vatarakta* are different according to site. In case of superficial *Vatarakta*, symptoms are *Kandu* (Pruritis), *Daha* (Burning), *Ruk* (Pain), *Ayaam* (Extension), *Toda* (Pricking pain), *Sphurana* (Throbbing pain), *Kunchana* (Contraction), *Tvak-Tamra-Shyava-Lohita-Varna* (Dusky red or coppery coloration of skin), *Bhanjana* (Cracking). In case of deep seated *Vatarakta*, symptoms are *Shvayathu* (Swelling), *Stabdhata* (Rigidity), *Kathinya* (Hardness), *Atiruk* (Agonizing pain), *Tvakashyavavarna*, *Tamravarna* (Dusky red or coppery coloration of skin), *Daha* (Burning), *Toda* (Pricking pain) *Sphurana* (Throbbing pain), *Paka* (Suppuration), *Granthi* (Glandular enlargement), *Sandhi-Asthi-Majjagat Chhedanavat Vedana* (Cutting pain in joints, bones and bone marrow), *Sandhi-Asthivakrata* (Deformity in joints and bones), *Khanjata* (Lameness), *Pangulya* (Paraplegia). Sometimes, both characters co-insides. *Vatarakta* exhibits features of Gouty Arthritis in modern medical science. Gouty Arthritis is a complex metabolic disorder of protein metabolism which results from deposition of monosodium urate monohydrate crystals in joint space causing inflammatory Arthritis. Reported prevalence of this Gouty Arthritis is 2.0 to 2.6 per 1000 patients, usually between the age group of 25-50 years. This article aims to study the better concept of *Vatarakta* in *Ayurveda* and its better co-relation as per medical science.

KEYWORDS: *Vatarakta*, *Kandu*, *Daha*, *Stabdhata*, Gouty arthritis.

## INTRODUCTION

*Vatarakta* is common presentation which is characterized by severe pain, tenderness, inflammation and burning sensation in the affected joints.<sup>[1]</sup> *Vatarakta* is a disease related with *Khavaigunya* found in *Raktavaha Srotas* which includes vitiated *Vata* and blood. Small joints of feet and hands are mainly affected in *Vatarakta*.<sup>[2]</sup> Purine is an important by-product of incomplete protein metabolism, leading into the excess of production of uric acid through their metabolic pathway viz. *denovo* and salvage pathways. Reduced renal clearance of uric acid and urate increases their level in circulation. These crystals get deposited in the joint space, triggering as inflammatory response. This response causes crippling disease called Gouty Arthritis.<sup>[3]</sup> The fundamental biochemical hallmark of gout is hyperuricemia which results from increased production or decreased excretion of uric acid or from a combination of the 2 processes.<sup>[4,5]</sup> Reported prevalence of this Gouty Arthritis is 2.0 to 2.6 per 1000 patients, usually between the age group of 25-50 years. The prevalence of gout is around 1% with a strong male predominance (10:1) primary gout is almost exclusively a male disease and most common cause of inflammatory arthritis in men over the age of 40 whereas secondary gout is due to renal impairment of drug

therapy mainly affecting people over the age of 65 and this form is usually seen in women.<sup>[6]</sup> In principle, *Vata* being pre dominant among the *Tridoshas*<sup>[7]</sup> has the potential to cause more serious and long term diseases than the other two. The classical texts lay down ample of importance to the functions and characteristics of *Vata*. At the same time, *Rakta* being the foremost body tissue also play an important role in sustaining the healthy life of the person. *Vatarakta* is an illness where both *Vata* and *Rakta* are afflicted by distinct etiological factors.<sup>[8]</sup> "*Vata Dushitam Raktam Yatra Roga Visheshah*". The disease characterized by the abnormally of *Rakta Dhatu* due to morbidity of *Vata Dosha* is called as *Vatarakta*.<sup>[9]</sup> *Nidans* like *Katu*, *Amla*, *Ushma*, *Vidahi Ahaaras*, *Gaja*, *Ushtra Yaana* is mentioned. When aggravated *Vata* is obstructed by aggravated *Rakta*, this obstructed *Vata* again vitiates the *Rakta*. This pathological stage is known as *Vatarakta*<sup>[10,11]</sup>

## Samprapti of Vatarakta

When *Rakta Dhatu* due to recurrent trauma or seasonal variation, gets vitiated and *Dosha* becomes enraged by the frequent use of *Kashaya*, *Katu*, *Tikta*, *Alpahar*, *Ruksha Ahara*, *Abhojana* or by regular riding of *Ashva*, *Ushtra* or *Yana*, *Ambukrida*, *Jalaplavana* or *Langhana*

and due to excessive walking during summer, or *Vega Nigraha*. The *Vayu* thus enraged and agitated enters into the blood, affect the entire blood, and leads to *Vatarakta*.<sup>[12-13]</sup>

### Types

Management of *Vyadhi* or *Roga* in the opinion of Ayurvedic scholars depends upon the variation of *Doshas*. As far as the varieties of *Vatarakta* is concerned Acharyas having categorized *Vatarakta* into two groups.

1. According to predominance of *Dosha*
2. According to site of origin

#### A. According to *Doshika* variation

According to *Doshika* variation Acharya Charaka has classified the disease *Vatarakta* in six categories: *Vata Pradhana Vatarakta*, *Pitta Pradhana Vatarakta*, *Kapha Pradhana Vatarakta*, *Rakta Pradhana Vatarakta*, *Dvandaj Vatarakta* & *Sannipataj Vatarakta*. Acharya Sushruta, Vagbhata, Madhavakara, Bhava Mishra, Sharangdhara have also described these varieties in their respective Samhitas.<sup>[14-19]</sup>

#### B. According to site of origin

According to site of origin Acharya Charaka, Chakrapani Datta, Yogratnakar have further classified *Vatarakta* as *Uttana Vatarakta* and *Gambhira Vatarakta*. Other Ayurvedic *Acharyas* have not mentioned these two varieties of *Vatarakta*. In spite of these one more variety of *Vatarakta* i.e. *Ubhaya Vatarakta* has been described by Acharya Charaka.

#### Sadhya-Asadhyata<sup>[20-26]</sup>

Regarding *Sadhya-Asadhyata* of *Vatarakta*, Acharya Charaka has pointed that if *Vatarakta* is newly originated and without complications originating from vitiation or provocation of a single *Dosha*, then it is *Sadhya* (Curable) condition. If it is caused by vitiation of *Tridosha* as well as associated with complications, it becomes *Asadhyata* or incurable. Further elaborating criteria of *Asadhyata*, Acharya Charaka has stated that the disease is *Asadhyata* when it is discharging profusely (*Samprasravi*) along with deranged colour (*Vivarnta*), stiffness (*Stabdhatta*), tumour (*Arbuda*), contracture (*Sankocha*) and damage the senses (*Indriya-Tapa*). Acharya Charaka has emphasised that besides these features is *Moha* (Mental confusion) is present. It makes the disease *Asadhyata*.

#### Concept of Gouty-Arthritis/ Hyperuricemia

Uric acid is the end-product of purine metabolism in humans. There is no universally accepted definition for hyperuricemia, but it is usually defined as serum urate concentration in excess of 6.8 mg/dL, which is the limit of urate solubility in serum. The role of high serum uric acid level as an independent risk factor for cardiovascular disease including stroke has been controversial. Several pathophysiological mechanisms through endothelial dysfunction, oxidative metabolism, platelet adhesiveness and aggregation, related to

hyperuricemia in cardiovascular disease have been suggested. According to a recent report 20, about 780,000 Americans experience a new or recurrent stroke each year, on average, one stroke every 40 seconds. Preliminary data from 2005 indicate that stroke accounted for about 1 of every 17 deaths in the United States. If asymptomatic hyperuricemia has a deleterious effect on serious morbidity and mortality related to stroke, hyperuricemia may become a new target for more comprehensive risk factor management in the primary prevention of stroke.<sup>[27]</sup>

#### Causes<sup>[28]</sup>

Hyperuricemia may be classified as primary or secondary depending on whether the cause is innate or is the result of an acquired disorder. However, it is more useful to classify hyperuricemia in relation to the underlying pathophysiology, i.e. whether it results from increased production, decreased excretion or a combination of the two.

#### Diagnosis

In most patients, the diagnosis of gout can be made with the presence of hyperuricemia and the presence of clinical features previously described for the diagnosis of gout, including recurrent attacks of acute arthritis, maximum inflammation developing within 1 day, attacks of mono arthritis, redness observed over joints, painful or swollen first metatarsophalangeal joint, unilateral first metatarsophalangeal joint attack, and unilateral tarsal joint attack.<sup>[29]</sup> Identification of urate crystals in tissue or synovial fluid of an inflamed joint is considered pathognomonic and the gold standard for diagnosis of gout. Examination of joint fluid is favorable for ruling out disorders that mimic gout, such as septic arthritis and acute calcium pyrophosphate crystal arthritis (pseudo gout), and having a clear conscience about planning the long-term urate-lowering therapy. However, synovial fluid sampling may not be feasible in all cases, and both false-positive and false-negative results may occur as well.<sup>[30,31]</sup> The difficulty of sampling of small joints and the need of an experienced operator for assessing the synovial fluid are other handicaps. Synovial fluid culture or Gram stain should always be undertaken if clinically septic arthritis is suspected, regardless of urate crystals in synovial fluid. Although hyperuricemia is regarded as a major risk factor for gout, a normal serum uric acid level does not exclude the diagnosis of gout.<sup>[32]</sup>

#### Treatment

Despite a sound understanding of the Samuel H. Poon, MD, Harald A. Hall, MD, and Bernard Zimmermann, MD synthetic and metabolic pathways that control serum uric acid levels, clinicians have been limited to a few urate lowering agents and one urate synthesis inhibitor since the development of allopurinol in 1956. Febuxostat became the second urate synthesis inhibitor when the Food and Drug Administration (FDA) approved it in early 2009. The role of febuxostat in the management of hyperuricemia and gout remains to be fully determined.

This review will discuss the traditional agents used for the lowering of serum uric acid and address the potential benefits febuxostat may offer in clinical practice.

### Sources of Serum Urate

Serum uric acid accumulates from the metabolism of purine nucleic acids which are derived either from cellular breakdown or directly from foods rich in purines such as red meats, beer, shellfish, and yeast extracts.<sup>[74]</sup> Catabolism of purines is mediated through a cascade of well-regulated enzymes that includes phosphoribosyl pyrophosphate synthetase (PRPS), hypoxanthine-guanine phosphoribosyl transferase (HRPT), and xanthine oxidase (XO).<sup>[33]</sup> A deficiency in HRPT or PRPS over-activity results in hyperuricemic syndromes like Lesch-Nyhan and Kelley-Seegmiller, with resultant gouty arthropathy in some patients. Unlike other animal species, humans and other primates do not express uricase, the enzyme which converts uric acid into the more soluble allantoin for excretion. Uric acid is therefore the end product in human purine catabolism and is ultimately excreted in urine and also, to a lesser proportion, in the stool. Drugs that inhibit xanthine oxidase and uricosuric agents that increase renal uric acid excretion are the cornerstone of therapy for hyperuricemia.

### DISCUSSION

*Vatarakta* is a disease of vitiated Vata along with vitiated *Rakta*. In Charaka Samhita it is mentioned that Vata due to subtleness (*Sukshmatva*) and pervasiveness (*Sarva-Sarvatva*) and *Rakta* due to liquidity (*Dravatva*) and flowing nature (*Saratva*), circulates through the body, through blood vessels (*Raktavaha-Srotasa*), gets obstructed in *Sandhies* (Joints) due to torsion nature of its course in joint, Vata and *Rakta* again agitated there. After *Sthan-Samshraya* or localization and in combination with Pitta etc. causes different type of *Shool* (Pain) according to the predominance of *Doshas*. The cardinal feature of *Vatarakta* is the sudden onset of joint pain (mostly in MTP joint) along with the inflammation of the joint.

Hyperuricemia, one of the major risk factors for the development of gout, is caused by an imbalance between the rates of production and excretion of uric acid. An excess of uric acid thus supersaturates the body fluids, leading to the deposition of monosodium urate crystals in tissues and joints, which in turn leads to the initiation of an acute attack of gout. Gout is a painful, occasionally debilitating disease and is divided into four clinical phases, namely asymptomatic hyperuricemia, acute gouty attack or recurrent gout, intercritical gout and chronic Tophaceous gout. Each of these phases has various possible treatments. NSAIDs, colchicine, corticosteroids and corticotrophin are used for the treatment of acute gout attacks in order to relieve pain. NSAIDs and colchicine are prescribed for the prophylaxis of gout. Allopurinol, probenecid and other uricosuric are used in long-term hyperuricemic therapy

in order to decrease serum uric acid levels and to prevent long-term damage to joints. Due to the untoward and, in some cases, potentially life-threatening side effects of the various gout therapies, there most definitely is a need for novel therapies to treat gout. Detailed patient evaluation forms the basis of a clinician's decision on the choice of therapy for gout patients. As such, more attention needs to be given to patient evaluation in order to personalise gout therapy for every patient. Emphasis should also be placed on the importance of diet and lifestyle modifications in addition to gout therapy so as to prevent gout and acute gout attacks.

### CONCLUSION

Concept of *Vatarakta* can be understood as a pathology occurring at the *Doshas* and *Dhatus*. Time has come upon to explore the principles of *Ayurveda* to serve the humanity. Advanced technology has made the life easy, which in turn has made the person lazy. He is running behind the success at the cost of his own health. Increasing stress and adoption of western culture has increased the incidences of life style disorders and metabolic disorders. *Vatarakta* (Gout) is one of the such disease. *Ayurveda* is the science of life which has the much potential to cure the *Vatarakta* (gout) from its root and also maintains the healthy life Later on. But this is possible only if we truly follow the *Chikitsa* sutras and *Pathya Apathya* mentioned in the *Ayurveda*. So there is great need of research on therapeutic principles of *Ayurveda*, on the formulations, on the therapeutic procedures like *Raktamokshana*, *Basti* etc.

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