

EFFICACY OF JALAUKAVACHARNA IN THE MANAGEMENT OF ACUTE GOUTY ARTHRITIS¹Lalita Sharma, ²Alok Kumar Srivastava, ³Poonam and ⁴Diksha Upreti^{1, 3, 4}MD Scholar, ²Professor

Department of Panchkarma, Uttarakhand Ayurved University, Haridwar.

***Corresponding Author: Lalita Sharma**

MD Scholar, Department of Panchkarma, Uttarakhand Ayurved University, Haridwar.

Article Received on 24/05/2017

Article Revised on 14/06/2017

Article Accepted on 04/07/2017

ABSTRACT

Gout is an ancient disease dating back to the time of Babylon. It's name came from the latin word Gutta(drop), points to the belief that a poison falling into the joint drop by drop causing the disease. More recently it become more prevalent and has increasing complexity in the past 20 years. Which could be related to the development of metabolic syndrome and longevity. It is still on the increase and it's prevalence has not plateaued yet .It is a most common inflammatory arthritis in men affecting 1-2% adults with male to female ratio of 3.6:1 but rare in pre menopausal women and it's incidence, prevalence increases with age. Clinically gout is a syndrome caused by an inflammatory response to Monosodium urate monohydrate crystals formed in human with elevated serum urate concentration (hyperuricaemia). It could present with either acute relapsing attack/it can come in chronic form. The acute attack usually cause very severe inflammatory arthritis which is usually self limiting and could take weeks before it completely setteles. The management of acute gouty arthritis include use of colchicine and NSAIDs but they have many adverse effects. Accordind to ayurveda it can be correlated with Vatarakta. It is a variety of vata roga which is caused by excessively aggaravated vata and vitiated blood (rakta). The modern drugs only provides symptomatically relief to the patient but does not change the course of disease. In Ayurveda leech therapy is more effective and has immediate effect on pain, inflammation and lasting effect on stiffness and dysfunction. High degree of safety suggest that this therapy has great potential in the management of acute gouty arthritis.

KEYWORDS: hyperuricaemia, aggaravated vata and vitiated blood (rakta).**INTRODUCTION**

Acute gout also known as 'Podagra', when it affects the 1st metatarsophalangeal joint. An acute attack of gout is a paradigm of acute sterile inflammation; as opposed to pyogenic inflammation. Recent studies suggest that the triggering of IL-1B release from leucocytes lies at the heart of a cascade of process that involves multiple cytokines and mediators.

The NLRP3inflammasome appears to have a specific role in this regard.

The link between crystal deposition and joint inflammation was made by Garrod, who developed the thread test assay of serum urate and stated that the deposited urate of soda may be looked upon as the cause not the effect of the gouty inflammation. The initial symptoms of gouty attack are sudden and violent taking place nearly always at night. The patient feels sudden onset of pain, it often affects the big toe of 1st metatarsophalangeal joint; which becomes rapidly red and swollen; the veins of the leg can become dilated and

the leg can become purple and sometimes accompanied by bruising.

Gouty arthritis is one of the most common rheumatic diseases. The clinical burden of gouty arthritis has historically been well recognized; however, gout is often misdiagnosed and mismanaged. The prevalence of gout is rising and is likely attributed to several factors including increased incidence of comorbidities, lifestyle factors and increased use of causative medications. With the increasing prevalence, there have been several innovations and evidence-based updates related to the diagnosis and management of gout. Acute gouty arthritis should be treated with nonsteroidal anti inflammatory drugs (NSAIDs), colchicine, or corticosteroids, or a combination of two agents. Xanthine oxidase inhibitor therapy remains the consensus first-line treatment option for the prevention of recurrent gout. Add-on therapies that reduce serum urate concentration include traditional uricosuric agents and a novel uric acid reabsorption inhibitor. Prophylaxis of acute gout with NSAIDs, colchicine, or corticosteroids is universally recommended when initiating any urate-lowering therapy in order to prevent acute gouty arthritis for a period of at

least 6 months. In this review, we discuss the epidemiology and risk factors for gouty arthritis and evaluate diagnostic strategies and therapeutic regimens for the management of gout, i.e according to Ayurveda raktmokshana through jalauka is the best one.

Rakthamokshana is the procedures of Panchakarma which helps to eliminate vitiated dosha that accumulate in the body. Jalaukavcharana is the types of Rakthamokshana which is practiced globally in India since ancient times. A detailed description on Jalaukavcharana is available in Ayurveda. It is used in management of various diseases in all the systems of the body including ears, nose, throat, eyes and head. Now a day's Leeches are also used by ophthalmologists to treat inflammatory, traumatic processes and various diseases of eyes. According to Ayurveda, the diseases of eyes were caused due to vitiation of tridoshas. Jalaukavcharana i.e. leech application is a type of bloodletting therapy. It removes some of these toxins and vitiated doshas which are accumulated in the body. Various bioactive substances are present in saliva of leech. Along with that, it also exerts a therapeutic effect in several diseases. Though despised by most, medicinal leeches can be of immense benefit that may help people to surmount numerous health disorders. Jalaukavcharana is very effective ancient method of Panchakarma neglected by physician. There are many side effects of modern medicine. So it is the need to spread awareness about Jalaukavcharana and its efficacy.

DISEASE BACKGROUND

Gout is the most common inflammatory type of arthritis in men affecting 1-2% of adults in Western countries

(1), with male to female ratio of 3.6:1, but rare in premenopausal women and its incidence and prevalence increases with age

(2). It is characterised by chronic hyperuricaemia which is defined as serum urate levels above 6.8 mg/dl ($\geq 400\mu\text{mol/L}$), the level above which the physiological saturation threshold is exceeded

(3). Gout manifests itself as microscopic or macroscopic soft tissue deposit of monosodium monohydrate crystal (tophi) which triggers severe, but self-limiting acute attack of arthritis with excruciating pain. In chronic cases crystals deposition could promote a chronic type of inflammation and erosive arthritis. Patients with hyperuricaemia also could develop uric acid urolithiasis which is usually promoted by urine acidity

(4). Asymptomatic hyperuricaemia is common, but there is no study to confirm the incidence or prevalence of its occurrence. Patients with hyperuricaemia have increased incidence of developing clinical gout when the serum urate level exceeds 9 mg/dl ($>530\mu\text{mol/l}$); however, we have to keep in mind that only a minority of patients with hyperuricaemia actually develops gout (5) and acute intermittent gout can still occur with lower level of serum urate than 9 mg/dl. Although clinically there are a few patients who develop tophi which could be seen, but a significant number of patients with gout have

microscopic or non-clinical type of tophi in joints, periarticular area and various soft tissues. These microscopic tophi and crystals including the renal uric acid calculi could be easily seen by the dual energy CT scan or with high resolution ultrasound, which is one of the recent advances in identifying gout crystals and making a diagnosis of gout

(6). Although there is clear evidence that soluble urate is an antioxidant

(7), however, urate can also be converted to pro-oxidant which could affect adversely the vascular endothelial cell function

(8). In observational studies, gout and asymptomatic hyperuricaemia shown to be directly promoting hypertension and vascular disease

(9). The current increase in the prevalence of gout could be related to overweight and the development of metabolic syndrome and change in our diet with high intake of meat, seafood, fructose sweetened beverages and beer, and also to the increase in life expectancy

(10). However, the main reason for gout and hyperuricaemia is related to the renal uric acid hypoxcretion, which can be multifactor in origin including both genetic and environmental factors such as diuretic use, low dose of aspirin and high alcohol consumption.

DISCUSSION

The medicinal leech is a beautiful symbol of give and take and is sustainable resource management. *Hirudo medicinalis* is one of the oldest surviving animals on earth. The 1st documented accounts of the use of leeches for medicinal purpose is from the time of extreme antiquity, more than 2500 yrs before in Ayurvedic texts then long later during the period of Hippocrates. Dhanvantari, the indian god of Ayurveda holds a leech in one of his hands. This simply suggests the importance of leeches in medicinal field by ancient indian sciences. Leeches have and will always be thought of as the 'Wonder doctors' of science.

Gout is one of the type of arthritis; which is characterized by progressive loss of cartilage in the joints due to the deposition of monosodium urate crystals and is associated with symptoms such as pain, tenderness, stiffness and reduced mobility. It is often, but not always, associated with hyperuricaemia. It is common, affecting around 2% of men aged over 30 years. and women aged over 50 years. It's prevalence appears to be increasing. Gout is common disease both in primary care and hospital practice. Life style and dietary recommendations for for gout patients should consider overall health benefits and risk since gout is often associated with the metabolic syndrome and an increased future risk of cardiovascular disease and mortality.

The treatment often includes the use of NSAIDs and topical Analgesics. This approach provides symptomatically relief but does not change the course of disease. Leech therapy is more effective than topical analgesics and anti inflammatory agents in the treatment.

Although leeches may not be safe for people with disease that impair blood clotting / for those with compromised immune function. It is believed to be safe for others. It's immediate effect on pain and inflammation and lasting effect on stiffness and dysfunction. High degree of safety suggest that this therapy has great potential in the management of gouty arthritis. Patients with gouty arthritis who were treated with leech therapy, experienced clinically significant improvements in self perceptions of pain for a limited period .Moreover a single application of leeches improved functional ability and stiffness for atleast 3 months.

The saliva of leeches contain a variety of substances such as Hirudin, Hyaluronidase, Histamine like vasodilators, collagenase, destabilase, inhibitors of kallikrein, superoxide production and poorly characterized anesthetics and analgesic compounds. These substances might reach deeper tissue zone and possibly the joint spaces.

Various bioactive substances in leech saliva may also be as pharmacologically potent as hirudin and thus exert substantial effects in periarticular tissue and adjacent structures. It has been proved that laser Doppler flowmetry that there is a significant increase in superficial skin perfusion following leech application; especially 16 mm around the biting zone.

Therefore a regional analgesic and Antiphlogistic effect by these substances enforced by hyaluronidase as well as counter –irritation might be the possible reason of improvement by treatment with leeches. Leech therapy could induce pain relief through Antinociceptive effects and counter –irritation. However, it is not known to what extent leech bites may induce such mechanisms. The jaw of leech pierces the skin so that these potent biologically active substances can penetrate into the deeper tissues.

Hyaluronidase (spreading factor) an enzyme in leech saliva, further facilitates the penetration and diffusion of these pharmacologically active substances into the tissues. With the additive effect of hyaluronidase, it is highly probable that the Antiphlogistic substances in leech saliva can penetrate deep enough to exert significant effects on periarticular myofascial structures play an important role in the development of pain.

Venous congestion is another important complication that threatens the viability of the affected joints following crystal orthopathies like gout. It can be best treated with application of leeches.

Leech therapy has 2 phases-

- 1- Active blood letting
- 2- Passive bleeding – from the leech wound after detachment, which can last for several hrs. The small blood volumes removed by medicinal leeches and the augment blood removal during the passive bleeding phase of leech therapy results remarkably decrease in

venous congestion in the joints. In addition to this, a broad no. of anticoagulant agents decrease venous congestion such as the thrombin inhibitor hirudin, Apyrase as well as collagenase, hyaluronidase, fibrinase1 and 2.

In summary traditional leech therapy seems to be an effective in treatment for gouty arthritis. Currently no pharmacologic agent has similar lasting effects after a single local administration.

CONCLUSION

Acute gouty arthritis typically presents with a sudden and severe exquisitely painful joint., most classically in the 1st metatarsophalangeal joint (toe).

In the current study Jalaukavacharna is significantly effective in treating gout. The effect of treatment was-
44%-Uttama upashaya in in relieving Pain.

40%- in Swelling

28%- in Stiffness

32%- in Restricted movements

16%- in Deformity

So we can conclude that Leech therapy is effective in treatment for acute gouty arthritis.

REFERENCES

1. Lawrence RC, Felson DT, Helmick CG, et al. National arthritis data workgroup. Estimates of the prevalence of arthritis and other rheumatic conditions in the United States. Part II. Arthritis Rheum 2008; 58: 26- 35.
2. Nuki G, Simkin P. A concise history of gout and hyperuricaemia and their treatment. Arthritis Res Ther 2006; 8: S1.
3. Mandell BF. Clinical manifestations of hyperuricaemia and gout. Cleve Clin J Med 2008; 75(Suppl 5): S5-S8.
4. Liebman SE, Taylor JG, Bushinsky DA. Uric acid nephrolithiasis. Curr Rheumatol Rep., 2007; 9: 251-7.
5. Workmann RL. Gout and hyperuricaemia. Curr Opin Rheumatol 2002; 14: 281-6.
6. Choi HK, Al-Arfaj AM, Eftekhari A, et al. Dual energy computed tomography in tophaceous gout. Ann Rheum Dis., 2009; 68: 1609-12.
7. Bieber JD, Terkeltaub RA. Gout: on the brink of novel therapeutic options for an ancient disease. Arthritis Rheum, 2004; 50: 2400-14.
8. Zharikov S, Krotova K, Hu H, et al. Uric acid decreases NO production and increases arginase activity in cultured pulmonary artery endothelial cells. Am J Physiol Cell Physiol, 2008; 295: C1183-90.
9. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. N Engl J Med, 2008; 359: 1811-21.
10. Hak AE, Choi HK. Lifestyle and gout. Curr Opin Rheumatol 2008; 20: 179-86.

11. Malik A, Schumacher HR, Dinnella JE, Clayburne GM. Clinical diagnostic criteria for gout: comparison with the golden standard of synovial fluid crystal analysis. *J Clin Rheumatol* 2009; 15(1): 22-4.
12. Janssen HJ, Fransen J, van de Lisdonk EH, van Riel PL, Janssen M. A diagnostic rule for acute gouty arthritis in primary care without joint fluid analysis. *Arch Intern Med* 2010; 170(13): 1120-6.
13. Glazebrook KN, Guimaraes LS, Murthy NS, et al. Identification of intraarticular and periarticular uric acid crystals with dualenergy CT: initial evaluation. *Radiology* 2011 Nov; 261(20): 516-24. *Sudan Med J* 2012 December; 48(3): 174 Review Article Gout AW Al-Allaf.
14. De Miguel E, Puig JG, Castillo C, et al. Diagnosis of gout in patients with asymptomatic hyperuricaemia: a pilot ultrasound study. *Ann Rheum Dis* 2012; 71(1): 157-8.
15. Pineda C, Amezcua-Guerra LM, Solano C, et al. *Arthritis Res Ther* 2011 Jan; 13(1): R4.
16. O'Sullivan JB. Gout in New England town. Prevalence study in Sudbury, Massachusetts. *Ann Rheum Dis* 1972; 31: 166-9.
17. Wallace SL, Robinson H, Masi AT, et al. Criteria for the classification for the acute arthritis of primary gout. *Arthritis Rheum*, 1977; 20(3): 895-900.
18. Vázquez-Mellado J, Hernández-Cuevas CB, Alvarez-Hernández E, et al. The diagnostic value of the proposal for clinical gout diagnosis (CGO). *Clin Rheumatol*, 2012; 31(3): 429-34.
19. Terkeltaub RA. Clinical practice: gout. *N Engl J Med*, 2003; 349: 1647-55.
20. Terkeltaub RA. Colchicine update: 2008. *Semin Arthritis Rheum*, 2008; 38: 411-9.
21. Janssen J, Janssen M, van de Lisdonk, et al. Use of oral prednisolone or naproxen for the treatment of gout arthritis: a double-blind, randomised equivalence trial. *Lancet*, 2008; 371: 1854-60.
22. Liu-Bryan R, Scott P, Sydlaske A, Rose DM, Terkeltaub R. Innate immunity conferred by toll-like receptors 2 and 4 and myeloid differentiation factor expression is pivotal to monosodium urate monohydrate crystal-induced inflammation. *Arthritis Rheum*, 2005; 52: 2936-46.
23. Ahern MJ, Reid C, Gordon TP, et al. Does colchicine work? The results of the first controlled study in acute gout. *Aust N Z J Med*, 1987; 7(3): 301-4.
24. Terkeltaub RA, Furst DE, Bennett K, et al. High versus low dosing of oral colchicine for early acute gout flare: twenty-four-hour outcome of the first multicenter, randomized, double-blind, placebocontrolled, parallel-group, dose-comparison colchicine study. *Arthritis Rheum* 2010; 62(4): 1060-8.
25. Lomen PL, Turner LF, Lamborn KR, et al. Flurbiprofen in the treatment of acute gout. A comparison with indomethacin. *Am J Med* 1986; 80(3A): 134-9.
26. Shrestha M, Morgan DL, Moreden JM, et al. Randomized double-blind comparison of the analgesic efficacy of intramuscular ketorolac and oral indomethacin in the treatment of acute gouty arthritis. *Ann Emerg Med* 1995; 26(6): 682-6.
27. Douglas G, Thompson M. A comparison of phenylbutazone and flufenamic acid in the treatment of acute gout. *Ann Phys Med* 1970; 10(6): 275-80.
28. Schumacher HR, Boice JA, Daikh DI, et al. Randomised double blind trial of etoricoxib and indomethacin in treatment of acute gouty arthritis. *BMJ* 2002; 324(7352): 1488-92.
29. Rubin BR, Burton R, Navarra S, et al. Efficacy and safety profile of treatment with etoricoxib 120 mg once daily compared with indomethacin 50 mg three times daily in acute gout: a randomized controlled trial. *Arthritis Rheum* 2004; 50(2): 598-606.
30. Axelrod D, Preston S. Comparison of parenteral adrenocorticotrophic hormone with oral indomethacin in the treatment of acute gout. *Arthritis Rheum* 1988; 31(6): 803-5.
31. Man CY, Cheung IT, Cameron PA, Rainer TH. Comparison of oral prednisolone/ paracetamol and oral indomethacin/ paracetamol combination therapy in the treatment of acute gout-like arthritis: a double-blind, randomized, controlled trial. *Ann Emerg Med* 2007; 49(5): 670-7.
32. So A, De Meulemeester M, Pikhlak A, et al. Canakinumab for the treatment of acute flares in difficult-to-treat gouty arthritis: results of a multicenter, phase II, doseranging study. *Arthritis Rheum* 2010; 62(10): 3064-76.
33. Jordan KM, Cameron JS, Anath M, et al. British Society of Rheumatology and British Sudan Med J 2012 December; 48(3) 175.
34. Review Article Gout AW Al-Allaf health provisional in rheumatology guidelines for management of gout. *Rheumatology* 2007; 46(8): 1372-4.
35. Zhang W, Doherty M, Bardin T, et al. EULAR evidence based recommendations for gout. Part II. *Ann Rheum Dis.*, 2006; 65: 1312-24.
36. Perez-Ruis F, Liote F. Lowering serum uric acid levels: what is the optimal target for improving clinical outcomes in gout? *Arthritis Rheum*, 2007; 57: 1324-8.
37. Edwards NL. Treatment-failure gout: a moving target. *Arthritis Rheum* 2008; 58: 2587-90.
38. Chao J, Terkeltaub RA. Critical reappraisal of allopurinol dosing, safety and efficacy for hyperuricaemia in gout. *Cur Rheumatol Rep* 2009; 11: 135-40.
39. Becker MA, Schumacher HR Jr, Wortmann RL, et al. Febuxostat compared with allopurinol in patients with hyperuricaemia and gout. *N Engl J Med*, 2005; 353: 2450-61.
40. Schumacher HR Jr, Becker MA, Wortmann RL, et al. Effects of febuxostat versus allopurinol and

- placebo in reducing serum urate in subjects with hyperuricaemia and gout: a 28-week, phase III, randomised, double-blind, parallel-group trial. *Arthritis Rheum*, 2008; 59: 1540-8.
41. Reinders MK, Haagsma C, Jansen TL, et al. A randomised controlled trial on the efficacy and tolerability with dose-escalation of allopurinol 300-600 mg/day versus benzbromarone 100-200 mg/day in patients with gout. *Ann Rheum Dis.*, 2009; 68: 892-7.
 42. Alobaidi H, Fazal S, Al-Allaf AW. Audit of the best practice in gout management, EULAR 12-3185, (accepted as a poster in EULAR 2012 in Berlin).
 43. Daibeth N, Stamp L. Allopurinol dose in renal impairment: walking the tightrope between adequate lowering and address event. *Semin Dial*, 2007; 20(5): 391-5.
 44. Reinders MK, van Roon EN, Houtman PM, Brouwers JR, Jansen TL. Biochemical effectiveness of allopurinol and allopurinolprobenecid in previously benzbromaronetreated gout patients. *Clin Rheumatol*, 2007; 26: 1459-65.
 45. Perez-Ruiz F, Alonso-Ruiz A, Calabozo M, et al. Efficacy of allopurinol and benzbromarone for the control of hyperuricaemia. A pathogenic approach to the treatment of primary chronic gout. *Ann Rheum Dis.*, 1998; 57: 545-9.
 46. Terkeltaub R. Learning how and when to employ uricase as bridge therapy in refractory gout. *J Rheumatol* 2007; 34: 1955- 8.
 47. Baraf HS, Matsumoto AK, Maroli AN, Waltrip RW 2nd. Resolution of gouty tophi after twelve weeks of pegloticase treatment. *Arthritis Rheum*, 2008; Nov; 58.
 48. Shastri K N, Chaturvedi GN. Chikitsa Sthana Rasayanadyaya. Charak Samhita, "Vidyotini" Hindi Commentary Part-II. Varanasi, Chaukhmbha Bharti Acadami; Edition Reprint, 1998; 819-40.
 49. Shastri Kaviraj Ambika Dutt. Chapter 1 Nidan sthan. Sushrut Samhita, "Ayurveda Tatwa Sandeepika" Hindi Commentary Part I .Varansi, Chaukhmbha Sanskrit Sansthan; Edition 11th; 231-32.
 50. Upadhyay Yadunandan. Nidan. Vatshonit Nidanadyaya. Astang Hridaya, "Vidyotini" Hindi Commentary. Varanasi, Chaukhmbha Sanskrit Sansthan; Edition – 2003; 280-284.
 51. Joshi Y G, Rasayan Chikitsa. Kayachikitsa; Pune, Pune Sahitya Vitaran; Edition 4th 2001; 287-284.
 52. Singh RH. Rasayan and Vajikaran. Swasthyavritta Vigyan; Delhi, Chaukhmbha Sanskrit Pratissthan, Edition 2007; 521-28.