



## CONSEQUENCES OF JALAUKAVACHARANA IN HISTOLOGICAL STUDY OF OSTEOARTHRITIS

**Dr. Abhilasha**

Assistant Professor, Faculty of Ayurvedic Science, Jayoti Vidyapeeth Women's University, Jaipur

There is mounting evidence that vascular pathology contributes to the onset of osteoarthritis, the most common form of joint disease (OA). Two probable causes include episodically decreased blood flow via microscopic capillaries in subchondral bone towards the ends of long bones and reduced interstitial fluid flow in subchondral bone. Blood flow can be restricted by venous obstruction and stasis, as well as the formation of microemboli in the subchondral veins. It is crucial to understand these potential aetiological factors so that more effective therapies to decrease the course of osteoarthritis may be created. In this context, Jalaukavacharana (Leech therapy), Leech therapy has great potential for the treatment of inflammatory, ischemic, and viral illnesses. The leech's saliva includes a number of physiologically and pharmacologically active compounds that have anti-coagulant, fibrinolytic, anti-platelet, anti-inflammatory, and anti-edema activities in the host's body. Arthritis, venous congestion, vascular diseases, abscess, and other problems of a similar condition.

**Keywords:** Jalaukavacharana, Leech therapy, Osteoarthritis, Synovial Fluid, Hirudin.

### Introduction

Osteoarthritis is the most prevalent joint disease in the world. Radiographic evidence of this disease is present in the majority of persons in Western nations by the age of 65, and in around 80% of those over the age of 75. Around 11% of adults over the age of 64 have symptomatic osteoarthritis of the knee.

The evidence for vascular pathology playing a role in the initiation and course of osteoarthritis which is the most prevalent joint disease. Although osteoarthritis is characterised by the gradual degeneration of articular cartilage. Subchondral cysts, sclerosis, and osteophyte formation are all symptoms of osteoarthritis. The symptoms of osteoarthritis, which include joint pain, stiffness, and articular cartilage loss, are caused by a variety of aetiologies. Increased weight, female sex, joint dysplasias, malalignment, and injury, among other risk factors for osteoarthritis, obviously contribute to the onset and development of this disorder<sup>1,2</sup>.

Nonsteroidal anti-inflammatory medicines (NSAIDs) are the most often prescribed drugs in modern medicine, despite the fact that they have several negative effects and are thus inappropriate for long-term use<sup>3</sup>. Raktamokshana<sup>4</sup>, often known as bloodletting, is an old and important parasurgical practise used in Ayurvedic medicine to treat a range of illnesses. Jalaukavacharana, also known as leech therapy, has gained popularity due to its medicinal benefits. Leech saliva includes a number of physiologically active components that have anti-inflammatory and analgesic properties. Charaka Samhita suggests If the symptoms increase after correct treatment of the vitiated Doshas, use Raktamokshana, which considers the role of the Rakta<sup>5</sup>. Nonsteroidal anti-



inflammatory medicines (NSAIDs) are the most widely prescribed drugs in contemporary medicine, although they have several adverse effects and are thus inappropriate for everyone. Jalaukavacharana (leech treatment) is a simple local Raktamokshana procedure for a painful joint. Given these facts, the purpose of this study is to investigate the histological perspective of osteoarthritis and to learn the scientific basis for the therapeutic impact of Jalaukavacharana in the management of joint pain.

### **Aim and Objectives:**

- To evaluate the histology of osteoarthritis.
- To evaluate the therapeutic effect of Jalaukavacharna on synovial fluid restoration.

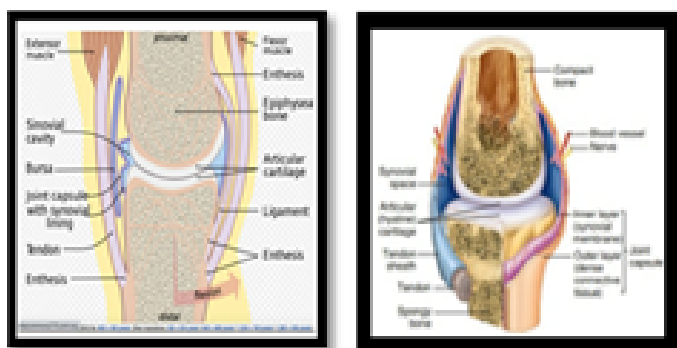
### **Histology of joints:**

Every step of bone production, repair, and metabolism involves the vasculature<sup>6</sup>. The blood supply to the bones serves both the marrow and the calcified bone tissue, and these two tissue types are functionally interrelated in terms of haemopoiesis, bone modelling, and remodelling<sup>7</sup>. The vascular supply of bone has many arterial inlets and venous outputs, with the four arterial inputs in the case of long bones being the nutritive artery, periosteal arteries, metaphyseal arteries, and epiphyseal arteries. Blood vessels are strategically placed to participate in the coupling of these processes, and blood vessels were discovered to be intimately associated with trabecular bone, particularly at sites of bone resorption. Long bone subchondral areas are extremely vascularized, indicating significant nutritional requirements<sup>8</sup>. Enhanced bone blood flow is also related with increased bone remodelling<sup>9</sup>. Compromised blood circulation in the subchondral bone, for whatever reason, may be damaging to the bone, but it also has implications for the avascular articular cartilage's integrity due to the subchondral bone's potential participation in feeding nutrients to the avascular articular cartilage.

### **Synovial Fluid:**

The inner membrane of synovial joints that secretes synovial fluid into the joint cavity is known as the synovial membrane<sup>10</sup>. Synovial fluid is a plasma ultrafiltrate containing proteins derived from blood plasma as well as proteins synthesised by cells in joint tissues<sup>11</sup>. The fluid comprises hyaluronan (secreted by synovial fibroblast-like cells), lubricin (secreted by articular cartilage surface chondrocytes), and interstitial fluid (filtered from blood plasma).<sup>12</sup> This fluid forms a thin layer on the cartilage surface (approximately 50 m) and seeps into microcavities and irregularities in the articular cartilage surface, occupying any empty space.<sup>13</sup>

The fluid in articular cartilage efficiently provides the synovial fluid reserve. During movement, the synovial fluid stored in the cartilage is mechanically forced out to maintain a fluid covering on the cartilage surface (so-called weeping lubrication).



**Fig1 -Vascularity of Synovial joint**

### **Disease Review:**

Osteoarthritis, often known as degenerative joint disease, is an illness that mostly affects cartilage. Cartilage is the slippery tissue that covers the ends of bones in a joint. With good cartilage, bones may slide over each other. It also helps with movement stress absorption. In osteoarthritis, the top layer of cartilage breaks down and wears away. This allows the bones beneath the cartilage to rub together. The rubbing causes joint discomfort, edoema, and range of motion loss. Over time, the joint may lose its normal shape. On the joint's borders, bone spurs might occur. Broken bone or cartilage fragments may float inside the joint, causing more pain and injury. The two types of osteoarthritis are primary (cataplexy) and secondary. Idiopathic osteoarthritis, the most common type of the illness, has no possible biomarker. Secondary osteoarthritis is pathologically identical to idiopathic osteoarthritis, but it has an underlying aetiology.

Bone remodelling occurs as a result of the coordinated action of osteoclasts and osteoblasts. The origin of bone remodelling is uncertain, however it appears that these sites are targeted, and the foundation for this targeting might be regions of decreased osteocyte viability<sup>14</sup>. Ischemia reduces osteocyte vitality, and osteocyte death leads to resorption of the dead bone segment, as established conclusively by intravital imaging<sup>15</sup>. By eliminating mechanical loads from bone and decreasing interstitial fluid flow, osteocyte hypoxia is quickly caused<sup>16-18</sup>. Serum-depleted osteocytes display a greater incidence of apoptosis in vitro, which can be somewhat eased by exposing these cells to fluid shear stress, which stimulates the production of the cell pro-survival protein<sup>19</sup>. As a result, osteocytes are capable of sensing mechanical signals in the form of fluid flow and, more importantly, appear to be dependent on fluid flow for life. Osteocyte death has also been linked to microdamage to the bone matrix. The first response to apoptotic osteocytes appears to be catabolic in bone, with osteoclast precursors quickly recruited and differentiated.



### The Efficacy of Leech Therapy on Osteoarthritis disease:

Ayurveda recommends Jalaukavacharana for sensitive or weak patients, female patients, old or too young individuals with Rakta-Pradoshaj Vikaras (blood originated diseases).<sup>20</sup> Jalaukavacharana is beneficial for disorders such as Vidradhi (abscess), Visarpa (inflammatory skin problems), Gulma (inflammatory condition of the abdomen), Pidika, Kustha, Charmadala (skin diseases), and others.<sup>21</sup> Leech therapy is frequently used in contemporary medicine to treat a variety of complex medical and surgical disorders, such as plastic surgery<sup>22</sup>, osteoarthritis (Osteoarthritis and Rheumatoid Arthritis)<sup>23-24</sup>, venous congestion<sup>25</sup>, vascular diseases, and thrombophlebitis etc.

**Table 1: Leech Saliva Components That Have an Impact In The Host's Body**

1.	Hirudin	Inhibits blood coagulation by binding to thrombin
2.	Hirustasin	Inhibits trypsin, kallikrein and chymotrypsin
3.	Calin	Inhibits blood coagulation and collagen- mediated platelet aggregation
4.	Destabilase	Dissolves fibrin and have thrombolytic effects
5.	Hyaluronidase	Antibiotic property, increases the permeability of the host skin
6.	Tryptase inhibitor	Inhibits proteolytic enzymes of host mast cells
7.	Factor Xa inhibitor	Inhibits the activity of coagulation factor Xa
8.	Acetylcholine	Vasodilator
9.	Chloromycetyn	Potent antibiotic
10.	Anesthetics substance	Anesthetic which is equally potent to morphine
11.	Histamine like substances	Vasodilator which increases the inflow of blood at the bite site

### Discussion:

Because existing therapies for knee osteoarthritis are limited<sup>26</sup>, new therapeutic strategies should be investigated. Leech treatment has not been researched in a modern scientific environment as a result of its extensive use throughout medical history<sup>27</sup>.

A multitude of mechanisms might explain the reported results. First, in addition to the thrombin inhibitor hirudin, other pharmacologically active chemicals in leech saliva have been found, including histamin-like vasodilators, inhibitors of kallikrein and tryptase, as well as other proteinase inhibitors and anaesthetics<sup>28</sup>.



Because of the simultaneous activation of another leech saliva component, hyaluronidase, these compounds may reach deeper tissue zones and perhaps the joint region. However, it is uncertain if direct contact with the cartilage and subchondral bone is required for pain alleviation in osteoarthritis. The many bioactive compounds discovered in leech saliva may be equally as potent as hirudin in terms of generating substantial effects on periarticular tissue and adjacent structures.

Second, nociceptive activation contributes to the onset of chronic pain. By acting on antinociceptive receptors, leech therapy may give pain relief. It's unlikely that a single reduction in nociceptive input would result in the claimed long-term consequences, such as improved joint function. Antiinflammatory substances like bdellins and eglins help to reduce inflammation, which reduces joint swelling. When the joint's inflammation decreases, so does the discomfort, and the joint's restriction.

Leech saliva includes hirudin, calin, and destabilase-like chemicals, which improve microcirculation by reducing blood viscosity. Corboxypeptidase An inhibitor increases the flow of blood to the bite site. Leech saliva also includes vasodilator-like histamine-like chemicals. In this way, the compounds found in leech saliva increase microcirculation, decrease inflammation, and alleviate stiffness and limitation of joint mobility.

### **Conclusion:**

In conclusion, conventional leech therapy for knee osteoarthritis appears to be an effective symptomatic treatment. The active compounds in leech saliva, as well as their local release (in synovial fluid), should be further explored. More investigation on the anti-inflammatory compounds present in leech saliva might lead to the development of new osteoarthritis medications. Based on the findings of the above review research, we can infer that leech treatment is beneficial in relieving pain, soreness, stiffness, crepitus, and edoema in osteoarthritis patients.

### **References:**

1. Harris WH. Etiology of osteoarthritis of the hip. Clin Orthop Relat Res 1986;213:20–33.
2. Buckwalter JA, Brown TD. Joint injury, repair, and remodeling: roles in post-traumatic osteoarthritis. Clin Orthop Relat Res 2004;423:7–16.
3. Lichtenstein DR, Syngal S, Wolfe MM. Nonsteroidal antiinflammatory drugs and the gastrointestinal tract. The double-edged sword. Arthritis Rheum 1995;38:5-18.
4. Shastri Ambika Dutt, editor. Sushruta Samhita Sutra Sthan vol-1. Jalaukavacharaniya adhyaya. Hindi Commentary. 14th Edition. Varanasi. Chaukambha Sanskrit Sansthan; 2003. p.43.
5. Gangasahay Pandey (Ed.), Commentarator of Charaka Samhita of Agnivesha- 1st volume, Sutra Sthan (24/11-16), Chaukumbha Sanskrit Sansthan, Varanasi (2006), pp. 444-445
6. Brandi ML, Collin-Osdoby P. Vascular biology and the skeleton. J Bone Miner Res 2006;21:183–92.



7. Compston JE. Bone marrow and bone: a functional unit. *J Endocrinol* 2002;173:387–94.
8. Imhof H, Breitenseher M, Kainberger F, Trattnig S. Degenerative joint disease: cartilage or vascular disease? *Skeletal Radiol* 1997;26:398–403.
9. Reeve J, Arlot M, Wootton R et al. Skeletal blood flow, iliac histomorphometry, and strontium kinetics in osteoporosis: a relationship between blood flow and corrected apposition rate. *J Clin Endocrinol Metab* 1988;66:1124–31.
10. Bay-Jensen, A. C.; Sand, J. M. B.; Genovese, F.; Siebuhr, A. S.; Nielsen, M. J.; Leeming, D. J.; Manon-Jensen, T.; Karsdal, M. A. (2016-01-01), Karsdal, Morten A. (ed.), "Chapter 31 - Structural Biomarkers", *Biochemistry of Collagens, Laminins and Elastin*, Academic Press, pp. 203–233, doi:10.1016/b978-0-12-809847-9.00031-3, ISBN 978-0-12-809847-9, retrieved 2020-10-18
11. Bennike, Tue; Ayturk, Ugur; Haslauer, Carla M.; Froehlich, John W.; Proffen, Benedikt L.; Barnaby, Omar; Birkelund, Svend; Murray, Martha M.; Warman, Matthew L. (2014-09-03).
12. Jay GD, Waller KA (2014). "The biology of lubricin: near frictionless joint motion". *Matrix Biology*. 39: 17– 24. doi:10.1016/j.matbio.2014.08.008. PMID 25172828.
13. Edwards, Jo, ed. (2000). "Normal Joint Structure". *Notes on Rheumatology*. University College London. Archived from the original on 19 November 2012. Retrieved 5 April 2013.
14. Noble B. Bone microdamage and cell apoptosis. *Eur Cell Mater* 2003;6:46–55.
15. Hsieh AS, Winet H, Bao JY, Glas H, Plenk H. Evidence for reperfusion injury in cortical bone as a function of crush injury ischemia duration: a rabbit bone chamber study. *Bone* 2001;28:94–103.
16. Dodd JS, Raleigh JA, Gross TS. Osteocyte hypoxia: a novel mechanotransduction pathway. *Am J Physiol* 1999;277:C598–602.
17. Gross TS, King KA, Rabaia NA, Pathare P, Srinivasan S. Upregulation of osteopontin by osteocytes deprived of mechanical loading or oxygen. *J Bone Miner Res* 2005;20:250–6.
18. Aguirre JI, Plotkin LI, Stewart SA et al. Osteocyte apoptosis is induced by weightlessness in mice and precedes osteoclast recruitment and bone loss. *J Bone Miner Res* 2006;21:605–15.
19. Bakker A, Klein-Nulend J, Burger E. Shear stress inhibits while disuse promotes osteocyte apoptosis. *Biochem Biophys Res Commun* 2004;320:1163–8.
20. Wolfe MM, Lichtenstein DR, Singh G. Gastrointestinal toxicity of nonsteroidal antiinflammatory drugs. *N Engl J Med* 1999;340:1888-99.
21. Sawyer RT. *Leech Biology and Behaviour*. Oxford: Oxford University Press; 1986.



22. Baskova IP, Khalil S, Nartikova VF, Paskhina TS. Inhibition of plasma kallikrein. Kininase and kinin-like activities of preparations from the medicinal leeches. *Thromb Res* 1992;67:721-30.
23. Eldor A, Orevi M, Rigbi M. The role of the leech in medical therapeutics. *Blood Rev* 1996;10:201-9.
24. Rigbi M, Levy H, Iraqi F, Teitelbaum M, Orevi M, Alajoutsijaarvi A, et al. The saliva of the medicinal leech *Hirudo medicinalis*—I. Biochemical characterization of the high molecular weight fraction. *Comp Biochem Physiol B* 1987;87:567-73.
25. Rigbi M, Levy H, Eldor A, Iraqi F, Teitelbaum M, Orevi M, et al. The saliva of the medicinal leech *Hirudo medicinalis*—II. Inhibition of platelet aggregation and of leukocyte activity and examination of reputed anaesthetic effects. *Comp Biochem Physiol C* 1987;88:95-8.
26. Claude A. Spreading properties and mucolytic activity of leech extracts. *Proc Soc Exp Biol Med* 1940;43:684.
27. Schaible HG, Ebersberger A, Von Banchet GS. Mechanisms of pain in arthritis. *Ann N Y Acad Sci* 2002;966:343- 54.
28. Michalsen A, Klotz S, Lüdtkke R, Moebus S, Spahn G, Dobos GJ. Effectiveness of leech therapy in osteoarthritis of the knee: A randomized, controlled trial. *Ann Intern Med* 2003;139:724-30.