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AYURVEDIC DRUGS IN MANAGEMENT OF INFLAMMATORY ARTHRITIS OF THE KNEE JOINT – A REVIEW

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ABSTRACT

Background: Inflammatory arthritis of the knee, including osteoarthritis and rheumatoid arthritis, arises from chronic synovial inflammation driven by cytokines such as IL-1β, TNF-α, and IL-6 (1). Ayurveda offers multiple botanicals with proven anti-inflammatory, antioxidant, and chondroprotective activities.

***Objective:** To review Ayurvedic drugs used in knee inflammatory arthritis, correlating their mechanisms with modern immunological pathways.*

***Methods:** Classical Ayurvedic texts and modern scientific literature indexed in Scopus, PubMed, and Web of Science were reviewed. Data on pharmacology, cytokine modulation, and joint-protective effects were extracted.*

***Results:** Thirty Ayurvedic botanicals demonstrated modulation of inflammatory mediators including NF-κB, COX-2, 5-LOX, TNF-α, IL-1β, MMP-3, and ROS. Many showed chondroprotective effects by inhibiting cartilage degradation enzymes and oxidative stress.*

***Conclusion:** Ayurvedic botanicals provide mechanistically supported anti-inflammatory and chondroprotective effects, suggesting usefulness as integrative therapies for knee inflammatory arthritis.*

INTRODUCTION

Inflammatory arthritis of the knee is characterized by synovial inflammation, pain, swelling, and progressive cartilage destruction (1). Modern pathology highlights the involvement of cytokines—TNF-α, IL-1β, IL-6—and enzymatic mediators such as COX-2, MMPs, and nitric oxide (2). Ayurveda describes Sandhivata and Amavata as conditions corresponding to degenerative and inflammatory arthritis (3). Herbal formulations, single-drug extracts, and rasayana agents have been traditionally used to modify inflammation, improve joint lubrication, and reduce pain (4). Recent advances in phytochemistry and molecular biology have elucidated mechanisms of Ayurvedic drugs that align with modern inflammatory pathways (5).

METHODS

A narrative review design was adopted. Ayurvedic classical texts including Charaka Samhita, Sushruta Samhita, Ashtanga Hridaya, and Nighantus were screened for drugs used in Sandhivata and Amavata (6). Electronic databases including Scopus, PubMed, Web of Science, and Google Scholar were searched using terms such as "Ayurveda", "anti-inflammatory herbs", "knee arthritis", "cytokine modulation", and "chondroprotection" (7). Studies examining phytochemical composition, anti-inflammatory pathways, enzymatic inhibition, and cytokine modulation were included. Data were extracted on the herb, active compound, and mechanism of action (8).

RESULTS

Thirty ayurvedic plants showing evidence for anti-inflammatory action were identified. Key mechanisms included: (a) NF-κB inhibition (Curcuma longa, Withania somnifera) (9); (b) suppression of COX-2 and 5-LOX enzymatic activity (Boswellia serrata, Zingiber officinale) (10); (c) reduction of IL-1β, TNF-α, and IL-6 (Tinospora cordifolia, Guduchi, Guggulu) (11); (d) antioxidant and ROS scavenging activity (Emblica officinalis, Asparagus racemosus) (12). Many herbs demonstrated chondroprotective effects via inhibition of matrix-degrading enzymes MMP-1, MMP-3, and MMP-13 (13).

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Table 1: Ayurvedic Drugs and Mechanisms in Inflammatory Knee Arthritis

Ayurvedic Drug	Primary Active Constituents	Mechanism of Action (Inflammatory Pathways)
Curcuma longa	Curcumin	NF-κB inhibition, ↓TNF-α, ↓IL-1β (14)
Boswellia serrata	AKBA	5-LOX inhibition, ↓Leukotrienes (15)
Withania somnifera	Withanolides	↓IL-6, ↓TNF-α, immunomodulation (16)
Tinospora cordifolia	Tinosporaside	Immunoregulation,↓pro-inflammatory cytokines (17)
Zingiber officinale	Gingerols	COX-2 and LOX inhibition (18)
Commiphora mukul	Guggulsterones	NF-κB downregulation (19)
Emblica officinalis	Vitamin C, tannins	Antioxidant, ↓ROS (20)
Asparagus racemosus	Shatavarin	Anti-inflammatory, antioxidant (21)
Plumbago zeylanica	Plumbagin	NF-κB suppression (22)
Ricinus communis	Ricinoleic acid	Anti-inflammatory, analgesic (23)
Piper longum	Piperine	↑Curcumin bioavailability, anti-inflammatory (24)
Piper nigrum	Piperine	COX inhibition (25)
Azadirachta indica	Nimbidin	↓IL-1β, ↓TNF-α (26)
Ocimum sanctum	Eugenol	COX-2 suppression (27)
Trigonella foenum-graecum	Saponins	Anti-inflammatory, antioxidant (28)
Cissus quadrangularis	Ketosterones	Bone/cartilage protection (29)
Moringa oleifera	Isothiocyanates	↓TNF-α and ROS (30)
Bacopa monnieri	Bacosides	Anti-oxidative neuro-modulatory (31)
Centella asiatica	Asiaticoside	Connective tissue repair (32)
Terminalia chebula	Chebulinic acid	Antioxidant, anti-inflammatory (33)
Terminalia bellirica	Lignans	Anti-inflammatory (34)
Tribulus terrestris	Saponins	Anti-inflammatory (35)
Glycyrrhiza glabra	Glycyrrhizin	COX-2 reduction (36)
Punica granatum	Ellagic acid	MMP-3 inhibition (37)
Berberis aristata	Berberine	↓IL-6, ↓TNF-α (38)
Saussurea lappa	Costunolide	NF-κB suppression (39)
Nerium indicum	Cardenolides	Anti-inflammatory modulation (40)
Coleus forskohlii	Forskolin	cAMP modulation, ↓inflammation (41)
Aloe vera	Aloin	↓IL-6, ↓TNF-α (42)
Vetiveria zizanioides	Sesquiterpenes	Antioxidant, anti-inflammatory (43)

DISCUSSION

The findings indicate that Ayurvedic botanicals act through multiple converging mechanisms. NF-κB inhibition, which suppresses transcription of pro-inflammatory cytokines, is a common pathway affected by Curcuma longa, Withania somnifera, and Guggulu (14,16,19). Inhibition of COX-2 and 5-LOX—key enzymes in prostaglandin and leukotriene synthesis—is achieved by Boswellia serrata, ginger, and holy basil (15,18,27). Many herbs possess chondroprotective properties through MMP suppression, antioxidant effects, and enhanced collagen synthesis (13). These mechanisms correspond with the modern understanding of synovial inflammation and cartilage breakdown, supporting their use as adjuncts in integrative arthritis management.

CONCLUSION

Ayurvedic botanicals possess diverse anti-inflammatory and chondroprotective actions validated by modern molecular studies. Their ability to modulate cytokines, oxidative stress, and joint degradation pathways supports their integration into the management of inflammatory knee arthritis. Further randomized controlled trials are needed.

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