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RESEARCH PAPER

Mechanistic insights into the anti-ulcerative effects of Avipattikar churna: a network pharmacology and experimental approach

Priya Shah[1,2] · K. Pundarikakshudu[2] · Vijay Kevlani[3] · Maitreyi Zaveri[1]

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Abstract

Introduction Avipattikar Churna is prescribed for gastric conditions in Ayurveda. The herbs present in churna have anti-inflammatory, antioxidant, and cytoprotective activities and the sugar part of churna is responsible for acid neutralization. We investigated the mechanism of action of Avipattikar churna against peptic ulcer using network pharmacology, in-vitro, ex-vivo and in vivo studies**.**

Methods Network pharmacology of churna was performed to identify the protein and pathways targeted by phytoconstituents of churna. In the phytochemical analysis the quantification of phenols, flavonoids, tannins, in vitro studies including acid neutralizing capacity, ex-vivo studies including proton pump inhibition and antioxidant activities of three extracts of churna (aqueous, alcoholic and hydro-alcoholic) and in vivo studies were performed.

Results Network analysis revealed Quercetin, Ellagic acid, Turpetholic acid, Copaene, and Eugenol having activity in peptic ulcer. The quantitative estimation of total phenols, flavonoids and tannins were found to be $12.467 \pm 0.273\%$ w/w, $4.734 \pm 0.091\%$ w/w, and $8.0368 \pm 0.138\%$ w/w respectively. The proton pump inhibition activity of all three extracts was concentration-dependent, with the hydroalcohol extract being the most effective at 250 ug/mL. Aqueous and hydroalcoholic extracts were found to improve levels of SOD, catalase and glutathione. Additionally, *in-vivo* studies revealed decrease in NFκB, TNF-α, and IL-6 post treatment. The ulcer protective effect was supported by histopathology results.

Conclusion From the experimental data we conclude that Avipattikar churna may act via various mechanisms including acid neutralization, proton pump inhibition and antioxidant activity. Further in vivo studies suggest that churna may suppress the NF-κB signaling pathway, which is associated with significant reductions in TNF-α and IL-6 levels, decreased inflammatory cell infiltration, promotion of ulcer healing, and induction of apoptosis.

Keywords Peptic ulcer · Antacid · Proton pump · Antioxidant · Network pharmacology · Inflammatory markers · Avipattikar churna

✉ Maitreyi Zaveri
khandharmaitreyi@gmail.com

Priya Shah
priyaashah86@yahoo.com

K. Pundarikakshudu
P_kilambi@yahoo.com

Vijay Kevlani
Vijaykvlani@gmail.com

[1] K. B. Institute of Pharmaceutical Education and Research, Kadi Sarva Vishwavidyalaya, Gandhinagar, Gujarat 382023, India

[2] Department of Pharmacognosy, L.J. Institute of Pharmacy, L J University, Ahmedabad, India

[3] School of Pharmacy, National Forensic Sciences University, Gandhinagar, Gujarat 382007, India

Abbreviations

ADMET	Absorption, distribution, metabolism, excretion, and toxicity
AKT1	Protein kinase B
ALC	Alcoholic
AQ	Aqeoues
ATP	Adenosine-5′-triphosphate
CACO-2	Colon adenocarcinoma
CHUK	Conserved helix-loop-helix ubiquitous kinase
CNSK2B	Casein kinase II subunit beta
DTNB	5, 5′-Dithiobis (2-nitrobenzoic acid)
EDTA	Ethylene diamine tetraacetic acid

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