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Formulation of plumbagin-loaded microemulsion: Evaluation of anti-rheumatoid efficacy in Wistar rat model

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ABSTRACT

Rheumatoid arthritis has become a common disease among the elderly. Plumbagin, a plant-derived chemical, has been shown to lower the levels of proinflammatory cytokines and interleukins linked to rheumatoid arthritis progression. The objective of the present research work was to assess the anti-arthritic activity of plumbagin-loaded microemulsion in CFA (Complete Freund's Adjuvant) induced arthritic rats. The plumbagin-loaded microemulsion was formulated with Captex 300 as oil phase, Tween 80, and Transcutol as surfactant mixture. The microemulsion system was analyzed for its physical appearance, pH, conductivity, zeta potential, globule size, compatibility, viscosity, drug content, and drug release rate. Formulation (F1P) with 0.5 % plumbagin, 8 % oil, 40 % S_{mix} and 52% water was selected as optimized formulation. The optimized formulation exhibited highest drug content (89.2%), *in vitro* release rate (87.36%) and lowest viscosity value (8.12 cP). Solubility of plumbagin in microemulsion was enhanced up to 24.38 times as compared to water. Upon intraperitoneal administration, plumbagin-loaded microemulsion remarkably reduced the concentration of proinflammatory cytokines (TNF-α, IL-6, IL-β, IL-10), liver marker enzymes (SGPT, SGOT, ALP) and restored the level of serum antioxidants (CAT, SOD, GSH) as compared to pure plumbagin in arthritic rat model. The data suggest an improved solubility of plumbagin by microemulsion system, which may lead to a reduced dose and better therapeutic outcomes.

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1. Introduction

Rheumatoid arthritis, a joint disorder, occurs when the immune system attacks or destroys its own cells and tissue. This malfunction results in chronic joint pain, boggy swelling, and muscular rigidity, eventually leading to bone erosion and joint deformity [1]. It is proposed that complex cell–cell interactions are responsible for the initiation and progression of the disease. It is assumed that antigen-presenting cells (APC) and CD^{4+} T cells interact together and initiate the reaction. APC carrying class II major histocompatibility complexes and peptide-based antigens attach to a specific type of receptors on the surface of T cells [2]. Patients with rheumatoid arthritis have been observed to lose bone integrity and become permanently disabled within a few years [3]. For the treatment of rheumatoid arthritis, nonsteroidal anti-inflammatory medications (NSAIDs) and corticosteroids are advised as first-line therapy, while disease-modifying anti-rheumatic medicines (DMARDs) are currently accessible as second-line therapy. Reportedly, they cause adverse side effects

Abbreviations: CFA, Complete Freund's Adjuvant; NSAIDs, Nonsteroidal anti-inflammatory drugs; DMARDs, Disease-modifying anti-rheumatic drugs; TEM, Transmission electron microscope; CAT, Catalase; SOD, Superoxide dismutase; GSH, Glutathione; MDA, Malondialdehyde; SGOT, Serum glutamic-oxaloacetic transaminase; SGPT, Serum glutamic-pyruvic transaminase; ALP, Alkaline phosphatase; PDI, Polydispersity index; TNF-α, Tumor necrosis factor-alpha; IL, Interleukin; APC, Antigen-presenting cells; CDR, Cumulative drug release.

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