ORIGINAL ARTICLE

Antihyperuricemic and xanthine oxidase inhibitory activities of Silymarin in a rat gout model

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Abstract

Introduction: Gout is a common metabolic defect spread around the world. It characterized by hyperuricemia, which resulting from the prolonged rise of uric acid (UA) levels in the blood, leading to increase the deposition of urate crystals in the joints and kidneys. The present study performed to investigate the efficacy of silymarin as antihyperuricemic agent. **Materials and Methods:** Enzyme assay was done using bovine milk xanthine oxidase (XO). The XO inhibitory activity *in vitro* was carried out using different doses of silymarin, and the degree of XO inhibition (XOI) was expressed as IC_{50}. The antihyperuricemic of silymarin was investigated in the potassium oxonate-induced hyperuricemic rat model for 7 consecutive days of oral treatment of 10, 25, and 50 mg/kg doses. **Results:** The study results revealed that the silymarin has a potent activity of XOI with IC_{50} = 5.84 µg/mL as compared to standard drug, allopurinol IC_{50} = 1.85 µg/mL. In addition, these results showed that all doses of silymarin were able to be significant reduced serum UA levels in the hyperuricemic rats. **Conclusion:** Silymarin showed a significant effect on lowering the level of UA in the evaluated model, and therefore, it may be a promising agent for treating gout because of the possession of an antihyperuricemic effect through the inhibitory activity of xanthine oxide.

Key words: Antihyperuricemic, gout, silymarin, xanthine oxidase

INTRODUCTION

Uric acid (UA) is the insoluble final product of purine digestion (DNA, RNA, and nucleotides). In the human body, nearly two-thirds of UA amount are the result of the degradation of endogenous purines, while the rest of the diet. Hyperuricemia means the precipitation of UA inside and around the joints and other tissues as a monosodium urate (MSU) crystal, and shedding of crystals into the synovial fluid generates a local inflammatory reaction. This caused joint inflammatory arthritis is termed gout. Gout is typically to a large degree painful, conventional therapy is nonsteroidal anti-inflammatory drug as a first remedy,[1,2] urate-lowering drugs such as allopurinol and probenecid.[3] Hyperuricemia is elevated in people with renal dysfunction, cardiovascular disease,[4] and hypertension.[5]

UA level in blood, furthermore, definitely increase with metabolic syndrome such as obesity, dyslipidemia, hyperglycemia, and insulin resistance.[6] UA was consumed as endogenous antioxidant for potent scavenger of reactive oxygen species and hydroxyl free radicals (OH). It is react with peroxy nitrile and stops nitric oxide synthase after was common believed it is metabolically inactive material, so UA acts as a pro-inflammatory and pro-antioxidant factor.[6,7] UA in blood indicator of pathologic circumstances damage by oxidation such as liver harm, hyperlipidemia, atherosclerosis, chronic heart failure, diabetes,[8] renal injury, fibrosis, and stimulating vascular smooth muscle proliferation.[9]

Silymarin is flavonoids compounds exist in *Silybum marianum*, as a chemical mixture of four isomers; silibin (major isomer), isosilbin, silycristin, and silydianin. This drug is an effective liver protective agent because it has a

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