Proc. Natl. Acad. Sci., India, Sect. B Biol. Sci.
https://doi.org/10.1007/s40011-018-1023-8

RESEARCH ARTICLE

In Vitro and In Vivo Study of *Argyreia speciosa* on Chronic Gastric Ulceration and Metabolic Studies

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Received: 26 May 2017 / Revised: 6 August 2018 / Accepted: 17 August 2018

Abstract Chronic gastric ulcer being the most prevalent gastrointestinal disorder is considered a major cause of gastric cancer. Large numbers of synthetic drugs ranging from proton-pump inhibitors and H_2 receptor antagonist to cytoprotective agents are available, but they have major side effects. Inadequacy of these synthetic drugs forces to develop new herbal-based drugs active against chronic gastric ulcer. In the present study, antiulcer activity of butanolic extract of *Argyreia speciosa* Linn. f., Sweet: Family Convolvulaceae (b-ASL) was investigated against acetic-acid-induced chronic gastric ulcer rat models. The effect of different dose of butanolic extract of *A. speciosa* was studied for antioxidant, anti-secretory, and cytoprotective activities. Extract administration significantly increased the gastric PGE_2 content. The further experiments including the in vitro metabolic stability and in vivo pharmacokinetics gave a clear indication that butanolic extract of *A. speciosa* (200 mg/kg) is an orally active and safe contender of antiulcer agents. It can be an important target for further studies under chronic ulcer management system.

Significance statement *Argyreia speciosa* significantly increased the gastric PGE2 content against ulcer. *In vitro* metabolic stability and *in vivo* pharmacokinetics gave a clear indication that *A. speciosa* is an orally active and safe contender of antiulcer agents.

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Keywords *Argyreia speciosa* · Chronic gastric ulcer · Flavonol · Metabolic stability

Introduction

Chronic gastric ulcer (CGU) is one of the commonest forms of stomach ulcer in developing countries. It comes under the most grave complications among ulcers. Reasons behind CGU are nonsteroidal anti-inflammatory drugs (NSAID), ethanol, stress, cigarette smoking, and *Helicobacter pylori* infection. These substances cause imbalance among factors like mucus bicarbonate, mucus secretion, blood flow, acid–pepsin secretion, cellular regeneration, prostaglandins, and epidermal growth factors [1]. This in turn leads to development of CGU. Reactive oxygen species are among the major culprits to be blamed for the formation of CGU. Free radicals liberated from the neutrophils and monocytes can cause oxidative damage to gastric mucosal cells by protein oxidation and lipid peroxidation [2, 3].

Chief therapeutic target for management of CGU is by regulating or neutralizing the gastric acid secretion [4, 5]. Anti-secretory medicine such as proton-pump inhibitors (omeprazole; OMZ), H_2 blockers (ranitidine, RND), and antacids turns out to be ineffective and also causes acid rebound when regularly used for longer times. This hypersecretion of acid after drug withdrawal/long-term use leads to high ulcer relapse rate. Extended treatment with these drugs cause serious side effects such as hypergastrinemia [6], osteoporosis [7], development of carcinoids in gastric mucosa, and increased risk of bacterial infection [8–10]. Clinical limitations of these synthetic drugs actuated the discovery of more effective and safer herbal

Published online: 29 August 2018