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Role of Some Medical Plants in Gastric Ulcer Healing

[1] Yasmin Sadek, [2] Salah Elballal, [3] Maysa Hanafy, [4] Anis Mohamed Zaid

[1, 3] Department of Pathology, Animal Health Research Institute, Dokky, Giza, Egypt
[2] Department of Pathology, Faculty of Veterinary Medicine, University of City, Egypt
[4] Department of Pathology, Faculty of Veterinary Medicine, University of Sadat City, Egypt

Corresponding Author: **Anis Mohamed Zaid**

Abstract

A significant portion of the gastrointestinal tract, especially in birds and mammals, is the stomach. Numerous variables, including inflammatory disorders and steroidal and non-steroidal anti-inflammatory medications used to treat a variety of illnesses, can result in gastric ulcers. Gastric ulcer aetiology is dependent on intricate interplay between aggressive and protective variables. Ulcers can develop when protective factors like sufficient mucosal blood flow, the mucosal bicarbonate barrier, endothelial cell regeneration, and ongoing prostaglandin production are outweighed by aggressive factors like gastric acid, pepsin, and reactive oxygen species. Herbs and plants are used in traditional medicine to cure and prevent a variety of gastrointestinal conditions, including peptic ulcers, without causing any negative side effects. including the anti-inflammatory activity of chamomile flavones and peptic ulcers without any negative side effects. It has been demonstrated that consuming foods high in polyphenols, such as olive oil, significantly reduces the production of hydroxyperoxides. Alpha-bisabolol reduces the proteolytic activity of pepsin by 50%. Some of these actions are crucial in the healing of the ulcers. One method in the prevention and treatment of gastric ulcers is to suppress the rate of gastric acid secretion. Raw honey has an inhibitory effect on gastric acid secretion. It is widely known that prostaglandin E2 protects the stomach mucosa from the damaging effects of gastric acid, and it has been demonstrated that rosemary extract increases prostaglandin E synthesis. Sesamol may have a protective effect on gastric mucosal damage in part by preventing mucosal ROS and the ensuing lipid peroxidation.

Keywords: Ulcer, Rosemary, Glutathione

Introduction

Food is stored and transported to the duodenum via the stomach, which makes up a significant portion of the gastrointestinal tract, especially in birds and mammals (**Treuting *et al.*, 2018**) [21]. It is made up of the fundic, pyloric, and cardiac areas, whereas the mammalian stomach is histologically divided into four layers: the muscularis, serosa, submucosa, and mucosa (**Mescher AL *et al.*, 2013**). Simple columnar epithelium and several gastric glands that connect to the stomach lumen via gastric pits make up the mucosa. Smooth muscles make up the muscularis layer, whilst connective tissue, blood, and lymph vessels make up the submucosa layer. The stomach's exterior is covered by a thin layer of serosa (**Bancroft JD *et al* 2008**).

Gastric ulcers are defined narrowly as mucosal defects that pierce the muscularis mucosa (**Stanton, M. *et al.*, 1989**) [18]. In actuality, however, gastric ulceration and gastrodudonal ulceration are structural, confined breaches in the surface of the gastrointestinal mucosa that characterise a clinical condition whose origin is probably complex and varies from case to case. Because acid pepsin bathes these ulcers, stomach or proximal duodenal ulcers are sometimes known as peptic ulcers (**Schaer, M *et al.*, 2003**).

It is crucial for any clinician to comprehend the aetiology and pathophysiology of gastrodudonal ulcers in order to treat them effectively and prevent them from developing in the first place. These ulcers can develop on their own, as a complication of numerous systemic diseases, or after different medications are administered to treat various diseases.

One well-known condition affecting the gastrointestinal tract is gastric mucosal injury. According to (**Karakaya *et al.*, 2009**) [8], the pathophysiology of stomach ulcers is based on intricate interplay between aggressive and protective elements. Ulcers

can develop when protective factors like sufficient mucosal blood flow, the mucosal bicarbonate barrier, endothelial cell regeneration, and ongoing prostaglandin production are outweighed by aggressive factors like gastric acid, pepsin, and reactive oxygen species (ROS) (**Li *et al*., 2006**) [11].

Furthermore, a number of apparently helpful treatments, such as dual antiplatelet therapy, low-dose aspirins, and non-steroidal anti-inflammatory medicines (NSAIDs), are significant risk factors for recurrent ulcers and associated problems (**Tang and Chan, 2012**) [20].

Inflammatory conditions, non-steroidal and steroidal (glucocorticoid) anti-inflammatory medications used to treat a variety of illnesses, primary and secondary stomach tumours, systemic diseases, and ingestion of toxic and other hazardous substances are all potential causes of gastric ulcers.

Numerous non-steroidal anti-inflammatory medications, such as aspirin, ibuprofen (**Godshalk, C. P. *et al*., 1992**), naproxen (**Malfertheiner P. *et al*., 2009**), and endomethacine (**Spee LA., 2010**), have been linked to ulcers in dogs, rodents, and horses (**Lanza, F. L. *et al*., 2009**) [10].

Gastric ulceration has also been linked to steroidal anti-inflammatory medications (**Grainek, I. M. *et al*., 2008**).

Furthermore, a number of apparently helpful treatments, such as dual antiplatelet therapy, low-dose aspirins, and non-steroidal anti-inflammatory medicines (NSAIDs), are significant risk factors for recurrent ulcers and associated problems (**Tang and Chan, 2012**) [20].

When administered orally at varying dosage rates, a lead salt mixture (Chloride Br, Sulphate) caused stomach ulcers (**Parrah *et al*., 2013**) [14].

As a potential strategy to prevent obesity in ovariectomised bitches, gastric ulceration has been documented as a complication after auto-transplantation of the ovaries to the portal vein drainage (**Davis MS *et al*., 2003**) [4].

Pathophysiology of gastric ulceration

The development of ulcers is caused by several pathophysiological processes.

However, suppression of the stomach mucosal barrier characteristics and promotion of gastric acid secretion are the common underlying pathophysiological mechanisms. Animals seldom have acid secretory problems, but they frequently have mucosal barrier disorders **(Parrah *et al*., 2013**) [14].

The multifactorial pathophysiological mechanism of gastric ulceration in dogs is caused by a variety of mechanisms, such as physical harm to the stomach mucosa, compromised mucosal defence, and chemical changes to the mucosa and its healing process (**Lacy ER., 1987**) [9].

Only severe trauma, such as gastric foreign substances or surgical implants, can cause ulcers to form from physical disturbance (**Ader P., 1979**) [1].

Numerous defence mechanisms that preserve and safeguard epithelial integrity are present in the gastric mucosa.The gastric epithelium layer is constantly renewing; deeper neck mucous cells take seven days to renew, while surface mucous cells do so in three days (**Strombeck, D. R. *et al*., 1990**) [19].

In order to cover the minor flaws in the stomach epithelial lining of the basal lamina, surface mucous cells ingrate within 30 to 60 minutes. To sustain protective and digesting activities, the mucosa receives at least 70% of the gastric blood flow **(Alan Barkun *et al*., 2010**) [3].

Maintaining sufficient blood flow during streaming may depend on physiological corticosteroid levels. Prostaglandin also promotes blood flow and raises the generation of mucus and bicarbonate. According to (**Duan SY *et al*. (2006**) [5], mucus has a high viscosity, is sticky, and easily forms a film that covers the epithelium.Pepsin cannot break down this mucous layer because it has selective permeability, retains alkaline fluid, and creates a buffer zone against an acidic or toxic environment. Oxyntic cells actively secrete bicarbonate ions into the luminal surface, which also serve as a chemical buffer.

There are numerous ways to compromise the mucosal defence system.The cycloxygenase pathway (COX-1 and COX-2), which produces prostaglandins from arachidonic acid, is typically inhibited by non-steroidal anti-inflammatory medications. The COX-1 pathway produces prostaglandins that are good for the stomach (**Parrah *et al*., 2013**) [14].

Without these helpful PGs, the generation of bicarbonate, gastrointestinal mucus, and blood flow are all decreased. Corticosteroids promote the development or duration of gastric ulcers by increasing the production of gastric acid and decreasing the creation of PG **(Rohrer CR *et al*., 1999**) [15].

In animals, shock decreases the amount of blood that reaches the stomach and causes acidosis due to inadequate perfusion, which both decrease the amount of bicarbonate ions that reach the surface cells.

Since ancient times, plants and herbs have been used in traditional medicine to prevent and treat a variety of gastrointestinal disorders, including peptic ulcers, without causing any negative side effects. Recognising the significance of plants in the development of novel and safer therapeutic agents, screening herbs for pharmacological activities and phytochemical constituents is one of the most active areas of research worldwide today (**Khair-ul-Bariyah, S. *et al*., 2012**).

Role of chamomile in gastric ulcer healing

Since ancient Egypt, Greece, and Rome, chamomile has been utilised in herbal treatment for thousands of years (Issac 1989). Many traditional, unani, and homoeopathic medical preparations contain chamomile as a key component (**Kumar *et al*., 2001**).

A significant category of cultivated medicinal herbs includes M. chamomilla. It includes a wide range of active and therapeutically intriguing chemical classes. Sesquiterpenes, flavonoids, coumarins, and polyacetylenes are among the most significant components of chamomile.

Chamomile extract contains eleven bioactive phenolic compounds, including herniarin and umbelliferone (coumarin), apigenin, apigenin-7-O-glucoside, luteolin and luteolin-7-O-glucoside (flavones), quercetin and rutin (flavonols), and naringenin (flavanone) (Gupta *et al*., 2010).

the flavones in chamomile that reduce inflammation. Pepsin's proteolytic activity is 50% reduced by alpha-bisabolol. According to earlier research, chamomile flower extract has a complicated impact on the stomach and duodenum's luminal and mucosal environments. While some of these behaviours are crucial for ulcer healing, others are crucial for preventing ulcer recurrence in the future. Acid secretion is directly impacted by chamomile aqueous extract, which also strengthens mucosal defence against harmful substances (**Rees, 1992**).

According to another studies, chamomile aqueous extract has anti-ulcerogenic properties when used alone or in combination with other plants (**Khayyal *et al*., 2001**).
A number of mineral elements, such as manganese and magnesium, as well as 1-2% volatile oils, such as α-bisabolol, α-bisabolol oxides A and B, and matricine found in chamomile flowers, may be linked to the anti-ulcer effect of CHAE (**McKay & Blumberg, 2006**).

Role of honey in gastric ulcer healing
The Holy Quran and other ancient religions have long praised honey. Because of its antibacterial qualities, honey has also shown promise in the treatment of burns, wounds, gastroenteritis, and skin ulcers. (**Ali AT and others, 2003**)
H-2 receptor antagonists, proton pump inhibitors (PPI), antacids, and anti-muscarinics are just a few of the medications that can be used to treat peptic ulcer disease; however, these treatments can have adverse effects on patients and do not always result in full recovery (**Meng J. *et al*. 2019**).
All religious texts have extensively covered honey, and it has been embraced by all ages, customs, and civilizations—ancient and contemporary. Ulcer animals in the control group had a gastric pH of 3.96 in an attempt to further investigate the potency of a safe and curative medication for the treatment of gastric ulcers utilising common and natural substances. However, when honey is administered, the pH of stomach contents rises gradually, depending on the dosage, to a maximum of 5.2460. stomach mucosal protection agents must be reinforced to combat ulcer-causing variables in order to prevent stomach ulcers (**Potrich FB. *et al*., 2010 and Mohamed AL *et al*., 2018**).
Therefore, reducing the rate of gastric acid secretion or neutralising it in the stomach mucosa is one method of preventing and treating gastric ulcers. revealed the inhibitory impact of raw honey on stomach acid secretion is comparable to that of cimetidine. The typical medication used in this study is cimetidine, which reduces gastric acid output by blocking intracellular adenylate cyclase, Na-K TPase, and parietal cell proton pumps and preventing histamine release due to H2 receptor inhibition (**R. Sathish *et al*., 2018**).
One of the primary ways that aspirin damages stomach cells is through ROS. However, animals given raw honey may have less oxidative damage to their stomachs if their MDA concentration significantly decreases (**Adefisayo MA. *et al*., 2018**).
Because MDA may be detected in bodily fluids, it is a valuable biomarker of lipid peroxidation (**Al-Wajeeh, NS. *et al*., 2017**).
Loss of membrane fluidity, disruption of ion transport and membrane integrity, and ultimately loss of cellular function are all consequences of lipid peroxidation (**Oluwole FS *et al*., 2016**). Raw honey is effective as a cytoprotective agent because it can help protect tissue by reducing the levels of MDA (**Hilary S *et al*., 2017**).

Role of olive oil in gastric ulcer healing
Polyphenols, a significant class of polar compounds with a variety of biochemical functions, including blocking and regulating radical reactions in the human body, are abundant in extra virgin olive oil. Free radicals raise the risk of chronic illnesses by oxidatively damaging macromolecules including DNA and lipids (**Nazzaro *et al*., 2019**).
It has been demonstrated that consuming foods high in polyphenols, such as olive oil, significantly reduces the production of hydroxyperoxides. Gastric ulcers have been linked to lipid peroxidation and reactive oxygen species (ROS) (**Palacios-Espinosa *et al*., 2014**).
The phenolic compounds in EVOO have been shown to have anti-inflammatory properties. (**Osman and others, 2017**) and antioxidant effects through nuclear factor erythroid-2-related factor activation, nitric oxide synthase expression suppression, and cyclooxygenase-2 inhibition (**Martinez-Huelamo M *et al*., 2017**).
According to reports, polyphenolic compounds may help prevent stomach ulcers because phenols may promote the production of PGE2 **(Alanko J *et al*., 1999**).
It is evident that polyphenols ameliorate the state of many oxidative stress biomarkers (**Williamson G *et al*., 2005**).
Their capacity to scavenge free radicals, break radical chain reactions, directly reduce peroxides, and stimulate the activities of antioxidative defence enzymes are just a few of the biological mechanisms through which these potential effects have been linked to their antioxidant properties (**Cook NC *et al*., 1996**).

Role of rosemary in gastric ulcer healing
Through a number of different ways, the rosemary extract was able to protect the gastrointestinal tract from the harm produced by ethanol. According to (**Queiroz *et al*., 2012**), rosemary extract is thought to have a strong anti-inflammatory impact on the stomach mucosa.
Furthermore, the protection of normal physiological levels of NO (nitric oxide) was another important mechanism exhibited by the components of the rosemary extract (this protective mechanism of the extract was detected by measuring the Nox (degradation products of NO).
By dilating the gastric blood vessels and increasing the supply of nutrients that support the growth of the cells that make up the granulation tissue—the first tissue to form in the regeneration process—the elevated levels of NO play a significant role in gastric protection.
Consequently, this helps the stomach mucosa repair (**Yang *et al*., 2000**).
The higher levels of reduced glutathione (GSH) linked to lower levels of oxidised glutathione demonstrated the antioxidant properties of the rosemary extract.
The studies showed that the components of the rosemary extract might function as antioxidants, oxidising themselves to lower the RS levels, which were raised as a result of ethanol-induced mucosal damage. The consumption of stomach GSH stores, which would have been converted to GSSG, was probably stopped by the antioxidant capacity of the rosemary extract. Furthermore, the components of the rosemary extract preserved normal catalase activity, a crucial antioxidant enzyme that breaks down hydrogen peroxide (H2O2) into oxygen and water.
However, due to an unidentified mechanism, neither ethanol nor rosemary extract were able to alter the physiological levels of SOD (super oxide dismutase) activity. The capacity of the components of the rosemary extract to shield cell membranes from RS attack further indicated the extract's protection against lipid peroxidation. (**Savegnago *et al*., 2006; Ineu *et al*., 2008**).
It is well known that prostaglandin E2 protects the stomach mucosa from the damaging effects of stomach acid. Because rosemary extract has been demonstrated to boost

prostaglandin E synthesis, it may be used to treat peptic ulcers. (**Y. Kimura *et al*., 1987**).

Role of sesame oil in gastric ulcer healing

Sesamol appears to have a protective effect on gastric mucosal damage, at least in part because it inhibits mucosal reactive nitrogen species and the ensuing lipid peroxidation.

Sesamol may have anti-inflammatory and antioxidative properties by inhibiting neutrophil activation and penetration into the stomach mucosa. The pathophysiology of NSAID-induced stomach inflammation and oxidative stress depends critically on neutrophil activation and infiltration (**Souza *et al*., 2008**). When neutrophils are activated, pro-inflammatory genes are expressed and pro-inflammatory mediators including TNF-α and IL-1ß are overproduced, which starts an inflammatory response (**Jaeschke & Hasegawa, 2006**).

Reactive nitrogen species, lipid peroxidation, and cell injury can all result from the overproduction of proinflammatory mediators, which can also enhance nitric oxide production (Hayes and McLellan, 1999).

Sesamol had a similar impact on the gastrointestinal mucosa of aspirin-treated rats, avoiding further stomach damage. This could be explained by a specific sesamol and aspirin combination that inhibits oxidative stress, mucosal inflammation, and neutrophil infiltration (**Hsu *et al*., 2009a**). Consuming sesame or sesame oil on a daily basis may help prevent damage to the stomach mucosa caused by long-term NSAID use. Along with other antioxidants including sesamin, sesamolin, and gamma-tocopheral, sesame lignan sesamol is a significant antioxidant found in sesame (**Tokusoglu *et al*., 2003**). They could all reduce oxidative stress on the stomach mucosa caused by NSAIDs. Consequently, it is hypothesised that consuming sesame or sesame oil on a daily basis could prevent damage to the stomach mucosa caused by NSAIDs. Additionally, pretreatment with sesame oil considerably reduced luminal haemorrhage and mucosal ulcer development caused by acidified ethanol. In an experimental rat model, sesame oil attenuates oxidative stress and gastrointestinal mucosal damage by reducing mucosal lipid peroxidation, glutathione, and nitric oxide generation (**Hsu *et al*., 2009b**).

Conclusion

Food is stored and transported to the duodenum via the stomach, which makes up a significant portion of the gastrointestinal tract, especially in birds and mammals, Gastric ulcers are defined narrowly as mucosal defects that pierce the muscularis mucosa, It is crucial for any clinician to comprehend the aetiology and pathophysiology of gastrodudonal ulcers in order to treat them effectively and prevent them from developing in the first place. These ulcers can develop on their own, as a complication of numerous systemic diseases, or after different medications are administered to treat various diseases. Corticosteroid increases gastric acid production and reduce PG's formation thereby promoting development or persistence of gastric ulcers. Since ancient times, plants and herbs have been used in traditional medicine to prevent and treat a variety of gastrointestinal disorders, including peptic ulcers, without causing any negative side effects. Recognising the significance of plants in the development of novel and safer therapeutic agents. Acid secretion is directly impacted by chamomile aqueous extract, which also strengthens mucosal defence against harmful substances. According to another studies, chamomile aqueous extract has anti-ulcerogenic properties when used alone or in combination with other plants, Loss of membrane fluidity, disruption of ion transport and membrane integrity, and ultimately loss of cellular function are all consequences of lipid peroxidation,Raw honey is effective as a cytoprotective agent because it can help protect tissue by reducing the levels of MDA. It is well known that prostaglandin E2 protects the stomach mucosa from the damaging effects of stomach acid. Because rosemary extract has been demonstrated to boost prostaglandin E synthesis, it may be used to treat peptic ulcers. Sesamol appears to have a protective effect on gastric mucosal damage, at least in part because it inhibits mucosal reactive nitrogen species and the ensuing lipid peroxidation.

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