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EDITORIAL

Is there still a role for sucralfate in the treatment of gastritis?

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Abstract

The endoscopic diagnosis of gastritis is usually made when a patient develops symptoms and undergoes an upper gastrointestinal endoscopy. There are often obvious aetiological causes such as smoking, alcohol *Helicobacter pylori* infection or drug treatment. Lifestyle changes can sometimes improve symptoms but often patients will be treated with a proton pump inhibitor. The stomach mucosa produces a protective mucous to prevent damage cause by gastric acid and exogenous agents can disrupt this layer. Repair of this protective layer can be enhanced by reduction in gastric acid secretion using H2 receptor antagonist or proton pump inhibitors or by cytoprotective drugs such as misoprostol, sucralfate, aluminium ions or bismuth subsalts. Sucralfate is a complex polymer which at a low pH changes its chemical configuration and binds to serum protein to form a protective layer protecting the mucosa against further injury. Cytoprotective drugs were the first line treatment for peptic disease including gastritis for many years but since the launch of cimetidine in 1976 and the subsequent launch of omeprazole in 1988, their use has slowly declined. First line treatment for patients with symptomatic gastritis after removal of potential causative factors is likely to be a proton pump inhibitor in 2019. This is despite the fact that there is some evidence that sucralfate is superior than a H2 receptor antagonist in the endoscopic healing rates in patients with gastritis. The logical treatment choice in patients with resistance symptoms is a combination of a proton pump inhibitor and sucralfate but evidence is lacking. Until such evidence is available In the meantime, we would suggest that there is a role for sucralfate in the treatment of intransigent gastritis and that mucosal protection should be considered even ahead of acid suppression given its favourable safety and toxicity profile.

Key words: Sucralfate; Enoscopy; Gastritis; Treatment; Anti-acid

Core tip: An endoscopic diagnosis of gastritis is commonly made. When patients have

significant symptoms associated with this finding and no other explanation can be found for their symptoms the first line treatment tends to be with a proton pump inhibitor. The combination of a proton pump inhibitor and sucralfate can however, be useful in the treatment of these patients when conventional treatment has failed and symptoms are severe.

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Gastritis is a common endoscopic diagnosis and can range from asymptomatic to severely symptomatic and be acute or chronic. Experts have long argued whether the endoscopic diagnosis of gastritis is in fact a gastropathy rather than a gastritis but in the present of an endoscopic diagnosis and symptoms treatment is usually offered. If we accept the endoscopic diagnosis of gastritis or gastropathy can cause symptoms then gastritis (as we will term in from here) is an inflammation, irritation or erosion of the gastric mucosa and can have a number of aetiological factors that include alcohol, smoking, various medications, *helicobacter pylori*, bile reflux or bacterial/viral infections. This condition should be differentiated from non-ulcer dyspepsia which has been extensively studied and is not associated with any endoscopic abnormality[1].

Management of gastritis can sometimes be difficult as it can be unresponsive to conventional therapy. Where there is an obvious aetiology for the gastritis removal of the cause is the first line option such as stopping smoking and reducing alcohol intake. This becomes more difficult when the cause is medication related and the medication in question is important to either quality or quantity of life. Under these circumstances, it usually comes down to a balance of risks. Treatment of *helicobacter pylori* infection where present often improves symptoms and there might be dietary changes that can be made to improve symptoms. Once these options have been, exhausted conventional medical treatment will be directed towards a reduction in acid secretion from the gastric mucosa, mucosal barrier protection or where gastritis is believed to be alkaline, gastric pro-kinetic agents.

The symptoms associated with gastritis are non-specific and it is therefore important to exclude any other cause for the patient's symptoms before assuming that they are related to gastritis. Symptoms include epigastric discomfort, nausea and early satiety. Patients will usually require a minimum of an endoscopy, *helicobacter pylori* test and an ultrasound scan. A careful drug, surgical and lifestyle history is necessary.

The gastric mucosa is protected by a layer of water-insoluble mucus gel that is approximately 180 micron thick[2]. This adherent mucus is the first line in mucosal defence against gastric acid in the lumen. Exogenous agents such as alcohol and certain drugs can disrupt the gel layer. The disruption of this layer is in part responsible for exposing the gastric mucosa to either acid or alkali and this can lead to gastritis. Repair of the gastric mucosal barrier can be facilitated by either anti-secretory agents such as H2 receptor antagonists or proton pump inhibitors or by cytoprotective drugs such as misoprostol, sucralfate, aluminium ions or bismuth subsalts.

Sucralfate is a complex polymer of sucrose with multiple substitutions of sulphate and aluminium salts. At a low pH it changes its chemical configuration, which allows it to bind to serum protein to form a protective layer over ulcerated areas. This protects the mucosa against further injury. Sucralfate also stimulates the synthesis and release of prostaglandins, epidermal growth factor and nitric oxide as well as improving gastric mucosal blood flow, bicarbonate secretion and mucus production[3]. Sucralfate is not absorbed systemically and therefore has a good safety and toxicity profile.

Prior to the introduction of H2 receptor antagonist and the subsequent introduction of proton pump inhibitors mucosal cytoprotective drugs were the first line treatment for peptic disease including gastritis. Since the launch of cimetidine in 1976 and the subsequent launch of omeprazole in 1988, the use of cytoprotective agents in the treatment of dyspepsia has slowly declined. First line treatment for patients with symptomatic gastritis after removal of potential causative factors is likely to be a proton pump inhibitor in 2019. This is despite the fact that there is some evidence that sucralfate is superior than a H2 receptor antagonist in the endoscopic healing rates in

patients with gastritis[4,5]. There is little evidence of superiority of proton pump inhibitors over sucralfate in the treatment of gastritis although the only evidence appears to be in post-cholecystectomy biliary gastritis[6]. There is no evidence of benefit from sucralfate alone or in combination in non-ulcer dyspepsia[1].

The logical treatment strategy in patients with symptomatic gastritis is the combination of acid suppression and mucosal protection. There is little or no literature that addresses this combination in any peptic disorder[7] and specifically the combination of sucralfate and a proton pump inhibitor does not seem to have been assessed in the treatment of resistant gastritis.

In our own practice, we have a selective group of patients, who have been extensively investigated to exclude other causes of their symptoms and who have symptoms resistant to conventional acid suppression. We have found anecdotal evidence of symptom relief with the combination of a proton pump inhibitor and sucralfate or in those with strong evidence of bile reflux and therefore alkaline gastritis from sucralfate alone. Our advice is always to take the proton pump inhibitor before the sucralfate and wait for an hour so that absorption is not affected. Our only concern is a lack of an evidence base to support this regime.

As this type of patient is common in both general practice and secondary care it would seem logical to design and conduct a randomised controlled trial to assess whether this approach is supported by scientific data.

In the meantime, we would suggest that there is a role for sucralfate in the treatment of intransigent gastritis and that mucosal protection should be considered even ahead of acid suppression given its favourable safety and toxicity profile[8].

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