Brief note on gastric mucosal ulcer.

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Description

Gastric mucosal concentrations of glucose, AMP, ADP and ATP were set on, also free fatty acids and cholesterol, after assessing variants in the phospholipid composition of the cell membrane. Helicobacter pylori cause type B gastritis. It seems strong association with the growth of gastric carcinoma. A plausible hypothesis for the lost link betwixt H. pylori infection, gastric carcinogenesis requires oxygen free radical-persuaded DNA damage.

An ulcer is a deep necrotic lesion penetrating along the entire thickness of the gastrointestinal mucosa and muscular is mucosae. Ulcer alleviating is a composite and tightly regulated process of filling the mucosal demerit with proliferating and migrating epithelial and conjugative tissue cells. This procedure covers the re-establishment of the constant surface epithelial layer, glandular epithelial structures, micro vessels and connective tissue inner the scar. Epithelial cells in the mucosa of the ulcer margin proliferate; migrate towards the granulation tissue to re-epithelialize the ulcer. Growth elements, being epidermal growth element (EGF), basic fibroblast growth element (bFGF), trefoil peptides (TP), platelet-derived growth element (PDGF), and other cytokines produced locally by reviving cells, command re-epithelialization and the redesign of glandular structures. These growth elements, most notably EGF, trigger epithelial cell proliferation across signal transduction pathways involving EGF-R- MAP (Erk1/Erk2) kinases. Granulation tissue that develops at the ulcer base consists of fibroblasts, macrophages and proliferating endothelial cells, which form micro vessels under the command of angiogenic growth elements. These growth elements [bFGF, vascular endothelial growth element (VEGF), and angiopoietins] promote angiogenesis—capillary vessel formation—thereby allowing for the design of microvasculature in the mucosal scar, which is essential for the supply of oxygen and nutrients to the healing site. The main trigger to activate expression of angiogenic growth elements and its receptors appears to be hypoxia. In the course of ulcer healing expression of growth, element genes are tightly regulated in a secular and spatially ordered way. A crude extract from Angelica sinensis (ASCE), which mainly consisted of polysaccharides, significantly promoted the migration and proliferation of normal gastric epithelial cells. These results hardly need that ASCE has a direct wound healing effect on the gastric mucosa. However, there is no report regarding the result of ASCE on gastric ulcer healing in animal models. ASCE promoted ulcer healing. The area of the ulcer was reduced. This was accompanied by notable growth in mucus synthesis when contrast with the control. Angiogenesis was inhibited by the treatment of ASCE. Cell proliferation, ODC and EGFR protein expression was not affected in this procedure.

A spontaneously perforating peptic ulcer located in a patch of heterotopic gastric mucosa in the upper portion of the esophagus led to the development of an esophagotracheal fistula. The histologic workup revealed, also to ulcer necroses and granulation tissue, glands of the fundic category, with head and parietal cells. On the base of histologic results, the fistula was successfully stopped with the help of a fibrin adhesive applied endoscopically. After the next session, the patient promptly becomes free of symptoms. Under medical cure with an HZ-receptor blocker, the patient has been totally symptom-free for 1 yr.

Angiogenesis is a fundamental process essential for the healing of substance stuff injury and ulcers because regeneration of blood microvessels is a critical requirement for oxygen and nutrient delivery to the healing site.

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