

# Vascular endothelial growth factor in portal hypertensive gastropathy essay



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Portal hypertensive gastropathy is increasingly being recognized as a major cause of anemia and bleeding apart from oesophageal varices. We know that, in portal hypertension, there is a relative hypoxia of the gastric antral and fundal mucosa, This mucosal ischemia causes increased risk of mucosal breakdown and thus leads to ulceration and in the setting of portal hypertension, it causes massive bleeding.

VEGF (vascular endothelial growth factor) has been proven to play a role in development gastric ulcers even in patients who are not having portal hypertensive, for example peptic ulcers. The data on increased VEGF in PHG is controversial with some authors reporting an increase, and others reporting decreased levels. In addition it is known that patients of PHT produce less gastric mucus secretion, which causes poor protection from acid environment of the stomach and leads to bleeding. The authors devised a study to assess factors such as role of VEGF, mucosal hexosamine and that of teprenone (a gastric mucosal protectant, which acts through a mechanism different from H1 blockade (like other antacids). It acts as a PGE2 (prostaglandin analogue).

An increase in the concentration of prostaglandin E2 allows rapid mucosal recovery of ulcers. Hexosamine is an indicator of mucosal protection. In this controlled study, the authors have made some significant observations. It has clearly been shown that VEGF and hexosamine are significantly raised in PHG patients. The gastric mucosal flow is also increased. Following administration of teprenone, the VEGF levels, hexosamine levels and the abnormally raised mucosal blood flow were reduced to optimum levels, allowing normalization of the gastric environment.

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This thus causes not only mucosal protection (with prevention of ulceration), but also allows developed ulcers to heal in a faster way. In summary, hypoxia which increases the gastric mucosal blood flow following induction of VEGF, and the poor mucus production may be restored to near normal levels with agents like teprenone which are PGE2 analogues. However the small size of the sample demands a larger study sample size.