

Pathogenesis in portal hypertensive gastropathy due to liver cirrhosis

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Abstrak

The recent advances of endoscopic examination had proven that source of upper gastrointestinal bleeding in liver cirrhosis is not always caused by esophageal varices rupture but also gastric mucosal lesion. The prevalence of gastric ulcer in patients with liver cirrhosis is higher than healthy individuals. Imbalance of defensive and aggressive factors of gastric mucosa may involve in development of portal hypertensive gastropathy (PHG). Several studies reported hemodynamic changes associated with portal hypertension causing decreased mucus layer thickness as one of mechanism of PHG. Other diabolic factors of PHG were hypoacidity, hypergastrinemia, reduced hexosamin concentration, mucus metabolic function associated with decreased prostaglandin E2, and increased nitric oxide which had caused mucus wall thickness changes. Gastric mucus damage induced by portal hypertension has important role in the pathogenesis of gastric ulcer in liver cirrhosis.