Portal Hypertensive Gastropathy: the Twilight Zone

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As one of the major complication of liver cirrhosis, portal hypertensive gastropathy (PHG) should be an interesting topic to study. Until now the exact mechanism on how this phenomenon happened is yet, not clear.[1] The occurrence of PHG is quite high in patient liver cirrhosis and some relationship occurred between PHG and portal hypertension. In liver cirrhosis patients, PHG may be closely associated with hepatic vein pressure gradient (HVPG).[2] However, PHG may not be directly associated with portal pressure because the mucosal damage not linearly correlated with portal hypertension, hence other mechanism involved in the etiopathogenesis of PHG.[1] In this regards, the most important factor related to PHG is the pressure in the splanchnic vasculature. Based on histopathology study in PHG, vascular congestion resulted from increased portal pressure reduced oxygen for gastric mucosa, hence exposed mucosal layer to irritants.[2] Moreover, the congestion somehow increased the production of nitric oxide, either through the shear stress locally or as the result of increase production of splanchnic vasculature due to portal hypertension.[3] This condition of increased oxidative stress ingastic mucosa mostly because of increase production of radical oxygen species and decrease production of tissue anti-oxidant such as glutathione synthetase and super oxide dismutase (SOD).[3] Our study (unpublished) along with other have shown that oxidative stress play a role in PHG and anti-oxidant reduced the gastric mucosal damage.[3] It is not clear whether gastric mucosal damage is due to increased susceptibility or related to increased of portal pressure. Patients with liver cirrhosis may not produce enough prostaglandin that needed by the mucosa as protection.[2] In this regard, mucosal damage in PHG may be the results of metabolic activity in local condition rather than merely an increased susceptibility of the mucosa for noxious agents. In line with this, the portal hypertension it self may be not sufficient enough to damage the gastric mucosa and liver dysfunction also play important part in pathogenesis of PHG. In PHG, acid secretion, both basal and stimulated secretion is reduced that may have relation with cirrhosis and portal hypertension.[2]

With all the complexity in the pathogenesis of PHG, changes in gastric pH and it consequences may not be easily understood. Rohmat et al in this publication showed that gastric pH increased along with severity of PHG.[4] They concluded that gastric pH is very likely an attempt to protect gastric mucosal damage. We know that gastric pH will be increased along with the decline in liver function. The mechanism regarding this condition not properly understood but may be related to the decline in the productions of proteins (enzymes or co-factors) that impaired along with declining of liver function. As such, the production of gastric acid also impaired. Whether increased of gastric pH will be protective to mucosa may be need further study. However, Rohmat et al were able to show that in liver cirrhosis patients, gastric pH can be increased without any intervention such as proton pump inhibitor.[4] This situation should also challenge the use of medications that can increase gastric pH. The use of proton pump inhibitor (PPI) that is quite frequently given to liver cirrhosis patients largely put into question. In PHG the use of PPI does not showed any efficacy and standard treatment will be the use of non-selective beta-blocker (NSB).[1,5] However, the efficacy of NSB in PHG only part of the solution because still significant numbers of patients have mucosal damage besides the usage of effective NSB. Changes in gastric pH as a consequence of to protect the gastric mucosa in PHG should be considered a passive event rather that active defense mechanism.

Several classifications for PHG have been proposed. Rohmat et al, determined the degree of mucosal damage of PHG based on McCormack study while the other one were proposed by the New Italian Endoscopy Club (NIEC) and Baveno III.[6] In NIEC classification the risk of bleeding was 3.5–31% in mild PHG and 38–62% in severe PHG.[1] Severe PHG reported by Rohmat et al in this publication was 61.3% that should makes every clinician aware that more than half of their patients have a major risk of bleeding because of PHG. Clinical presentation of PHG commonly not in

the form of profuse bleeding but in chronic blood loss and because of it, in liver cirrhosis patients, severe PHG should be consider as one condition related to anemia, if any. Treatment with NSB should be evaluated with cautioned. [5]

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