十二指腸潰瘍發生及復發相關因子的研究

Studies in Factors Related to Developm ent and Recurrence of Duodenal Ulcer

中文摘要

本研究在探討造成十二指腸潰瘍發生以及復發之相關因子來設計實驗。我們利用電子內視鏡進行上消化道檢查，證實是否有十二指腸潰瘍的存在。在胃液酸度與十二指腸潰瘍的關係，我們抽取少許胃液，進行胃液酸度 pH 值檢測，利用 student's T test 檢定後比較潰瘍患者及非潰瘍患者之胃液酸度 pH 值，結果顯示潰瘍患者有較低的胃液酸度（P ＜ 0.05）。在幽門螺旋桿菌與十二指腸潰瘍的關係，我們採取少許切片做 CLO test 以及 H&E stain 來證實幽門螺旋桿菌存在與否。結果顯示十二指腸潰瘍患者有較高的幽門螺旋桿菌感染率（P ＜ 0.05）。在自由基與十二指腸潰瘍的關係，我們分析急性潰瘍患者與非潰瘍受檢者，十二指腸黏膜中 Cu/Zn-SOD 的含量，結果顯示在潰瘍活動期，十二指腸黏膜有較高含量的 Cu/Zn-SOD（P ＜ 0.05）。在前列腺素 E2 與十二指腸潰瘍的關係，我們分析急性潰瘍患者與非潰瘍受檢者，胃黏膜組織中前列腺素 E2 的含量，結果顯示十二指腸潰瘍患者與非潰瘍受檢者之胃黏膜組織中，前列腺素 E2 的含量，在統計學上，並無有意義的差異 (P ＞ 0.05)。在十二指腸潰瘍復發的因子上，我們討論再生黏膜的組織成熟度並研究十二指腸黏膜之胃上皮化生及球部變形與潰瘍復發的相關性，結果顯示再生黏膜的組織成熟度愈差者與胃上皮化生的程度及球部變形的程度愈嚴重者，有較高的潰瘍復發率。我們的結論是十二指腸潰瘍發生的因子是多樣性，包括攻擊因子如胃酸、幽門螺旋桿菌及自由基等過多有關，但是防禦因子如前列腺素 E2 的含量在十二指腸潰瘍上似乎不若胃潰瘍來得明確。此外更重要的是在十二指腸潰瘍復發的研究上，發現十二指腸再生黏膜的組織成熟度與十二指腸球部變形的程度及胃上皮化生的程度確實與十二指腸潰瘍的復發有密切的相關性，而根除幽門螺旋桿菌，可能可以減少十二指腸球部變形的程度以及胃上皮化生的程度，因而避免潰瘍的一再復發。

英文摘要

A study has been conducted by our institute to investigate factors related to development and recurrence of duodenal ulcer.TV-endoscopy was used in this study to confirm the existence of duodenal ulcer (DU) . For studying the relationship between gastric acidity and duodenal ulcer,

small amount of gastric juice were aspirated from patients with or without duodenal ulcer during endoscopic examination to check the intragastric pH using pH meter. The result showed that there was significant lower gastric pH in duodenal ulcer patients ($P < 0.05$) as compared with patients without duodenal ulcer. For studying the infection rate of H. pylori in patients with duodenal ulcer, gastric specimens were taken during endoscopy to perform CLO test and H & E staining. The infection rate of H. pylori were 89.4% in DU patients and 50.8% in non-ulcer patients. The result showed that DU patients have a higher infection rate ($P < 0.05$) as compared with patients without duodenal ulcer. Regarding the relationship between free radical and DU, we analyzed Cu/Zn-SOD content in duodenal mucosa of acute DU patients and non-ulcer patients. The result showed a significant higher content of Cu/Zn-SOD in DU patients ($P < 0.05$). In conjunction to PGE2 and DU, we analyzed the PGE2 level of gastric mucosa in both acute DU patients and non-ulcer patients and the result showed that there was no significant difference between patients with and without duodenal ulcer ($P > 0.05$). For further studying the recurrence of duodenal ulcer, we discussed histological maturity of regenerating mucosa and studied the correlation among the grade of gastric metaplasia, deformity of duodenal bulb and the recurrence of DU. The result showed that the less histological maturity of regenerating mucosa, the higher the level of gastric metaplasia and the more severe the deformity of duodenal bulb the patient have, the higher rate of DU recurrence the patient would have. In conclusion, there were multiple factors related to development and recurrence of DU, they consist of aggressive factors such as gastric acid, H. pylori and excessive free radicals and protective factors, such as poor maturity of regeneration mucosa. However, we found that PGE2 do not play an important role in the DU development. Because the correlation among histological maturity of regenerating mucosa, the grade of gastric metaplasia, severity of deformity of duodenal bulb and ulcer recurrence are highly associated. We suppose that eradication of H. pylori could reduce the level of gastric metaplasia and deformity of duodenal bulb and thus prevent the recurrence of DU.