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C-GH-6

A Study on the Effect of COX-2 Inhibitors on Gastric Mucosal Prostaglandin Synthesis

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Introduction: Recent evidence suggests that celecoxib, a specific inhibitor of cyclo-oxygenase-2 (COX-2) enzyme, causes significantly less peptic ulcers than conventional non-steroidal anti-inflammatory drugs (NSAIDs). We aimed to study the long-term effect of celecoxib on the synthesis of prostaglandin in human gastric mucosa.

Method: Forty patients who developed peptic ulcers while receiving NSAIDs were recruited into the study. After the ulcer was healed with anti-ulcer drugs and *Helicobacter pylori* (if present) was eradicated, patients were randomised to receive celecoxib 100 mg twice daily or naproxen 750 mg daily plus lansoprazole 30 mg daily for 3 months. Two gastric biopsies were taken from baseline and at 3 months to assess gastric prostaglandin production (PGE₂) using an ELISA method.

Results: One patient in the celecoxib group and no patient in the naproxen plus lansoprazole group developed recurrence of gastric ulcer. Naproxen caused a significant reduction of PGE₂ synthesis at month 3 by 48% ($p < 0.05$ compared with baseline). No significant reduction in the gastric mucosal PGE₂ was detected in patients receiving celecoxib (6% vs baseline, $p > 0.05$).

Conclusion: Long-term administration of celecoxib did not inhibit the gastric mucosal prostaglandin production. This may explain the reduced ulcerogenic effect of COX-2 specific inhibitor.

C-GH-7

Prognostic Factors in Severe Exacerbation of Chronic Hepatitis B

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Background: Prognostic factors for severe exacerbation of chronic hepatitis B are important in the decision for early liver transplantation. The aims of this study were to identify the prognostic factors for patients with severe exacerbation and to examine the role of precore and core promoter mutations.

Methods: Forty-seven patients with severe exacerbation were recruited. The liver biochemistry, viral serology, HBV DNA levels, and outcome were monitored. Precore and core promoter mutations were determined in 24 of these patients, and compared with 96 with mild exacerbation and 96 without exacerbation.

Results: Fifteen patients (36.2%) died or had liver transplantation. Prothrombin time of >30 seconds on admission was the single most important factor for poor prognosis, with 85.7% of patients having adverse outcome. Other factors associated with adverse outcome were low albumin, high bilirubin and low platelet count on admission; peak bilirubin levels, peak prothrombin time, long time to reach the peak prothrombin time during subsequent monitoring; and the presence of encephalopathy or ascites. There was no difference in the frequency of precore mutation in patients with severe exacerbation (62.5%), with mild exacerbation (54.2%) and without exacerbation (44.8%). There was a significantly lower prevalence of core promoter mutants in patients with severe exacerbation (50%) when compared to that with mild exacerbation (81.3%, $p = 0.004$).

Conclusions: According to the identified prognostic factors, patients with severe exacerbation could be categorized into a high-risk group in whom early liver transplantation might improve outcome.