



Hong Kong Society of Gastroenterology

Newsletter Volume 1 Issue 1

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News...

Health Sciences & Nursing Studies (Diploma in Gastroenterology Nursing)

The Society has organised a Diploma Course in Gastroenterology Nursing jointly with the School of Professional and Continuing Education, (HKU) and Hong Kong Society of Endoscopy Nurses in July 1998.

The course aimed at benefiting registered nurses who are interested in gastroenterology nursing, especially if they are currently working in Endoscopy Units and Medical/Surgical Units with patients who have gastroenterological disorders. The course has given nurses in-depth knowledge of gastroenterology and the opportunity to observe/function in gastroenterology units in different hospitals.

Message from Dr. W.M. Hui, President

Hello! Happy New Year.

On behalf of the council members, I am pleased to announce the publication of the Newsletter of the Hong Kong Society of Gastroenterology. I would like to take this opportunity to thank our fellows and members for their continued support of the functions of the Society. The Society is 18 years old. In 1999, the Society will have another active year. Our scientific programme is growing bigger. Updates and position papers on important and controversial issues in gastroenterology will be published regularly. We hope to promote training and research activities among our fellows and members.

Through these, we hope to upkeep and upgrade our standard of gastroenterology. These can only be achieved with your active participation. We hope to see as many of you at our scientific meetings. Do contribute, interact and participant.

Research Committee

The Society would play an active role in promoting research in gastroenterology. To achieve this, the Council has set up a research committee. The aim of the committee is to identify controversial or unresolved problems in gastroenterology, especially those topics which are prevalent, or getting more prevalent in Hong Kong. Relevant questions will then be addressed and members in the field will be invited to participate in the project. Through the conduct of the research and its results, we hope to improve the standard of gastroenterology, especially in the era of evidence based clinical gastroenterology. These studies will be funded by the Society. The first area that we have identified is colorectal cancer screening as the "incidence" of colorectal cancer is increasing in Hong Kong. We hope that members will play an active role in making these projects a success.

Scholarship Selection Committee

The Society is pleased to announce that the Scholarship Selection Committee, which has set up recently, has established a scholarship for Hospital Authority medical staff and members of the Society. The objective of the Scholarship is to facilitate trainees in gastroenterology (physicians, surgeons, radiologists, pathologists) of the Hospital Authority or the Society to undertake overseas training in gastroenterology. Interested parties are welcome to lodge application in writing enclosing the curriculum vitae. The deadline of application is on April 30, 1999. For any enquiries, please contact Miss Clare Wong (Tel: 2855-3354, Fax: 2816-2863 or E-mail: mcwong@hkucc.hku.hk)

Francis K.L. Chan

Dr. Francis Chan obtained the Bachelor degree of Medicine with honours in the Chinese University of Hong Kong in 1988. He received medical training in the Department of Medicine, Prince of Wales Hospital after graduation. In 1994, he went to Canada as a Croucher Foundation research fellow to study bile formation in experimental liver transplantation. He was appointed as Associate Professor in the Department of Medicine & Therapeutics, the Chinese University of Hong Kong in 1997 and obtained the degree of Doctor of Medicine in 1998. His research interests include bile salt kinetics in experimental liver transplantation, NSAID-associated peptic ulcer disease, *Helicobacter pylori* and gastric carcinogenesis.

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Risk factors in NSAID-associated ulcer disease

Non-steroidal anti-inflammatory drug (NSAID) is the one of the most commonly consumed drugs worldwide. Patients who take NSAIDs have a four- to six-fold increased risk of developing peptic ulcer disease. Furthermore, up to 60% of NSAID-induced ulcer complications can occur without any antecedent symptoms (1). Certain factors are associated with increased risk of ulcer disease in users of NSAIDs. These include:

- Past history of peptic ulcer or ulcer complication
- Advanced age
- Major comorbid illness
- Concomitant steroid or anticoagulant therapy
- High dose or multiple NSAIDs
- NSAIDs with high gastric toxicity
- Duration of NSAID treatment
- Helicobacter pylori* infection

Among these risk factors, past history of upper gastrointestinal bleeding is the single most important predictor of recurrent ulcer hemorrhage in NSAID users (relative risk and 95% confidence interval = 13.5 [10.3-17.7]) (2). Controversies exist as to whether the elderly have increased susceptibility to the adverse effects of NSAIDs. The elderly population is the biggest consumer of NSAIDs. It follows that they bear the greatest burden of disease. Furthermore, the increased risk may be a reflection of the known increased propensity to peptic ulcer complications in older patients (3).

Several comparative studies have observed that different NSAIDs possess different ulcerogenic potentials, ranging from ibuprofen (low risk) to piroxicam (high risk) (4). Others argue that the different ulcerogenic risk is more related to the pattern of prescribing than the ulcerogenic potential of these drugs. It was found that piroxicam was often prescribed to older patients with disabling arthritis, whereas ibuprofen was frequently given at lower doses to younger patients with mild arthritis (5). Previously it was thought that different formulations or routes of administration might influence the gastric toxicity of NSAIDs. Now it is apparent that the frequency of ulcers is similar with oral, parenteral, or rectal administration of NSAIDs. Neither prodrugs nor enteric-coated NSAIDs can reduce the frequency of gastroduodenal ulcerations.

Epidemiological studies reveal that the risk of ulcer complications is highest in the first month of NSAID therapy (6), suggesting that these early complications might occur in those with preexisting *H. pylori*-associated gastritis or peptic ulcers. In a prospective randomized study of *H. pylori* eradication before starting NSAID treatment, prophylactic cure of *H. pylori* infection markedly reduces the 8-week cumulative rate of NSAID-induced ulcer (7).

Prevention of NSAID-associated ulcer complications

Many ulcer complications are related to injudicious use of NSAIDs. Clinicians should avoid prescribing NSAIDs unnecessarily to high-risk subjects. Strategies to prevent adverse gastrointestinal side effects associated with NSAIDs include:

- Avoid/eliminate risk factors
- Cotherapy with anti-ulcer drugs in high-risk groups
- Eradication of *H. pylori* infection
- Use safer NSAIDs?

In a large-scale randomized controlled trial of misoprostol for the prevention of serious ulcer complications associated with NSAIDs, cotherapy with misoprostol has been shown to reduce the rate of ulcer complications. Nevertheless, over 4000 patients were treated only to prevent 17 episodes of ulcer complications. Twenty-five percent of patients dropped the therapy because of gastrointestinal upset (8). Therefore, the use of misoprostol should be limited to high-risk NSAID users only. Other endoscopic studies show that omeprazole and high-dose H₂-blocker can prevent erosions and ulcers associated with NSAIDs (9-11). Whether these endoscopic observations can be translated to clinical benefits remain uncertain. Two large-scale studies reported higher therapeutic success rates in NSAID-associated gastroduodenal lesions and symptoms with omeprazole compared with ranitidine and misoprostol (10,11). However, the results were based on comparison between standard dose of omeprazole and low dose of ranitidine and misoprostol. It remains unknown whether proton pump inhibitor is really superior to H₂-blocker and misoprostol if the latter drugs are given at full doses.

Although the role of *H. pylori* in NSAID-associated ulcer disease remains controversial, it is probably advisable to eradicate *H. pylori* in infected subjects. This is because clinically one can hardly distinguish pure NSAID ulcers from *H. pylori* ulcers in chronic NSAID users. A recent study suggests that eradication of *H. pylori* in patients on long-term NSAIDs might impair healing of gastric ulcers and did not affect the rate of ulcer relapse detected by screening endoscopy (12). However, in another prospective randomized study of *H. pylori* infected patients presenting with NSAID-associated bleeding gastric ulcers, curing the infection did not have any adverse effect on ulcer healing (13). On the other hand, long-term use of NSAIDs is associated with a continuous risk of ulcer complications. In patients with past history of NSAID-associated bleeding ulcers, eradication of *H. pylori* alone cannot entirely eliminate the ulcerogenic risk of NSAIDs (14). Therefore, cotherapy with anti-ulcer drugs should be considered in *high-risk* patients (i.e., prior ulcer complications) requiring long-term NSAIDs.

A new class of NSAIDs, namely, selective COX-2 inhibitor, has been the focus of research recently. The selective activity against cyclooxygenase-2 (COX-2) maintains the anti-inflammatory (therapeutic) activity of the drug via COX-2 inhibition while the adverse gastric effect (COX-1 inhibition) is spared. Certain drugs show increased in vitro selectivity against COX-2 e.g. nabumetone, etodolac, meloxicam and nimesulide. Although early clinical data suggest that these drugs might have lower gastric toxicity than conventional NSAIDs, the rate of serious gastrointestinal events is not reduced (15). Other new agents with more specific inhibition against COX-2 are under investigation e.g. celecoxib, MK-966. More data are needed to establish the safety and efficacy of these specific COX-2 inhibitors.

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multifactorial, which may include dietary factors, environmental factors, bacterial and viral infections. The role of *Helicobacter pylori* infection in gastric carcinogenesis is important. Several large clinical trials are trying to address the causal relationship between *Helicobacter pylori* infection and gastric cancer.

Epidemiology

Until recently, gastric cancer was the most frequently diagnosed cancer in the world. In the mid 1980s, gastric cancer was displaced by lung cancer and became the second commonest malignancy. The highest incidence in Asia are found in Japan, China and South Korea. Areas in Asia with low incidence include India, Pakistan and Thailand. There is no consistent pattern. Within a country, there are also considerable differences in incidence. Several parts of China have a very high incidence rate, like Changle, Fujian while in some parts the incidence is quite low (1). In small countries like Japan, there is also considerable variation in the incidence and mortality rate (2). The incidence rate of gastric cancer increases with age. The male to female ratio in incidence rate is usually around 1.5 - 3.0 worldwide, while in China it varies from 1.6 - 3.9 (3).

Gastric cancer has been associated with low socio-economic status, based on family income (4), education (5), or occupation (6). Generally, gastric cancer risk of the lower social class was up to two times greater than that of the upper social class. Of interest is that *Helicobacter pylori* infection is also associated with low socio-economic status (7). Familial studies have found that the risk of relatives of cases in developing gastric cancer is increased two- to three-fold (8). However, since family members usually share the same environment and has similar socio-economic status, it is difficult to exclude environmental factors while interpreting these findings.

Helicobacter pylori

There is now increasing evidence from the epidemiological studies that *Helicobacter pylori* carriers are at significantly higher risk of developing gastric cancer. The best evidence was derived from the three prospective epidemiological studies (9-11) which estimated *Helicobacter pylori* carriers to have a 2.8-6 times increased risk of developing gastric cancer over mean follow up periods of 6-16 years when compared with their *Helicobacter pylori* negative counterparts. The overall mean risk was calculated to be 3.8 in a meta-analysis of these three studies (12). There was a significant trend towards an increased odds ratio with increased length of follow-up (12). Six out of nine case-control studies from various parts of the world demonstrated significantly increased risk of developing gastric cancer in *Helicobacter pylori* carriers while the other three did not show any differences (13-21). Differential *Helicobacter pylori* infection rates have been observed in contrasting gastric cancer areas in different geographical regions; with the majority of the studies demonstrating a significant correlation between the two (22-30). Two previous studies from China (26,29) demonstrated an unequivocal association between gastric cancer mortality rates and *Helicobacter pylori* infection rates. Our previous study also showed that the prevalence of *H. pylori* infection was higher in Changle than in Hong Kong, where the gastric cancer mortality in Changle was about ten times that of Hong Kong (31). The odds ratio increased in young patients. Kikuchi et al (32) showed in a case-controlled study that at average age of 34 years, the odds ratio for *H. pylori* carriers to have gastric cancer was 13.3.

There were reports that gastric cancer mortality rates bear an inverse relationship to duodenal ulcer disease rates and duodenal ulcer: gastric ulcer ratios both in China and in other countries (33-35). It remains a puzzle why the same organism can cause two diseases and yet one disease seems to protect against the other. The pathogenesis of the two diseases seems so different that they are likely to involve a mutually exclusive

Helicobacter pylori and Gastric Cancer



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Introduction

In 1996, approximately one million new cases of gastric cancer were found worldwide. Currently, it is the fourth leading cause of cancer death in Hong Kong. Like most malignancies, the etiology of gastric cancer is still unclear. It is generally agreed that the pathogenesis is

pathway. Some other factors besides *H. pylori* are likely to be involved in the determination of ulcer or cancer formation.

In the gastric environment, *H. pylori* infection leads to changes that are important in the pathogenesis of gastric cancer, including the vitamin C level in gastric juice, reactive oxygen metabolites, and epithelial cell proliferation (36-38). Two host factors received much attention recently. One study demonstrated a significant association between the prevalence of the HLA-DQ5 genotype and *H. pylori* infection with accompanying atrophic gastritis or intestinal metaplasia (39). Study by McColl et al has shown that a small group of subjects appear to show more profound suppression of gastric acidity as a result of *H. pylori* infection which may lead to development of severe gastric atrophy and further hypochlorhydria. (40) One of the environmental factors that received attention is salt consumption. The Intersalt study and a Japanese study demonstrated an association between salt intake and gastric cancer risk. (41,42)

Looking at the organism itself, specific pathogenic *H. pylori* strains have been incriminated as the culprits. Blaser et al. demonstrated that CagA+ *H. pylori* patients were at a higher risk of developing intestinal metaplasia and gastric cancer (43). Moreover, CagA producing *H. pylori* strains were consistently found to be more prevalent in patients with peptic ulceration (44,45) and to a certain extent in patients with gastric cancer (46,47) in clinical studies. In a recent controlled study, Parsonnet et al. (47) demonstrated that subjects infected with the CagA+ strains had a 5.8 folds increase in developing gastric cancer when compared to the uninfected subjects. On the other hand, compared with the latter, those who had been infected by the CagA- strains only had marginally and insignificantly increased risk of developing gastric cancer. Two other case-controlled studies performed in areas with high gastric cancer and high background prevalence of CagA+ strains (48,49), however, did not support this finding. Thus, it remains controversial if CagA+ strains are relevant in causing gastric cancer.

There is sufficient evidence to show the relationship between *H. pylori* infection and gastric cancer. In 1994, the Working Group Meeting of the International Agency for Research on Cancer, in affiliation with the World Health Organisation, concluded that *H. pylori* was carcinogenic to humans (Group 1 carcinogen)(50).

Prevention of gastric cancer

The initial chemoprevention trials were all based on high risk subjects, those with precancerous lesions in the stomach. The end point was regression or progression of the precancerous lesions. There were three, large scale chemoprevention trials in this category. The one in Colombia was designed to treat the subjects (chronic atrophic gastritis, intestinal metaplasia, dysplasia) with anti-*H. pylori* therapy and was then randomized to treat with β -carotene and vitamin C or placebo. Another study in Venezuela randomized the subjects (chronic atrophic gastritis, intestinal metaplasia, dysplasia) to receive vitamin C + β -carotene + vitamin E or placebo. The European Cancer Prevention- Intestinal Metaplasia Study Group randomised the subjects with intestinal metaplasia to receive anti-*H. pylori* treatment, and then vitamin C supplementation or placebo(51).

Another approach in chemoprevention trials was based on asymptomatic subjects to see if eradication of *H. pylori* could reduce the overall incidence of gastric cancer. The end point will be cancer incidence in the cohort. There are four chemoprevention trials looking at this in asymptomatic *H. pylori* carriers. Our group started the study in 1994 (31). A total of 1600 asymptomatic subjects in Changle, Fujian, China were randomized to receive anti-*H. pylori* treatment or placebo in 1994 without micronutrient supplements. The effect on cancer incidence and change in precancerous lesions will be investigated by a second

upper endoscopy around 1999 in Changle. Two other studies in Shangdong and one in Japan used a similar design aiming at cancer prevention by eradication of *H. pylori* (52,53).

There are now at least two more intervention trials looking at precancerous lesions and one more trial on looking at cancer incidence as an end point. With the results of all these trials in the coming few years, we may be able to offer some strategies for prevention of the world's second most common cancer.

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Forthcoming Events

Joint HKSGE, HKSDE and HKSCP Scientific Meeting

Date : March 27, 1999

Place: Grand Ballroom, Lower Level 1, Kowloon Shangri-La Hotel, 64 Mody Road, Kowloon

For further information

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Scientific Symposium on Current Practice of Hepato-biliary and Pancreatic Surgery

Date : April 12, 1999

Place: Furama Hotel, 1 Connaught Road Central, Hong Kong

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Digestive Disease Week 1999

Date : May 16-21, 1999

Place: Orlando, Florida, USA

For further information

Tel: 1 609 848 1000 Fax: 1 609 853 5991 E-mail: ddw@slackinc.com

United European Gastroenterology Week

Date : November 13-17, 1999

Place: Rome, Italy

For further information

Scientific Secretariat

Tel : 39 06 445 2872

Fax : 39 06 4938 5147

E-mail: roma99chair@uni.net

Conference Organiser

Tel : 39 06 328121

Fax : 39 06 324 0143

E-mail: ega_uegw99@uni.net

14th International Workshop on Therapeutic Endoscopy

Date : December 7-9, 1999

Place: Endoscopy Centre, Prince of Wales Hospital

For further information

Tel: 2632-2233 Fax: 2635-0075

E-mail: info@hksde.org

AGA-GAT Joint Meeting

Date : December 12-16, 1999

Place: Chiangmai, Thailand

For further information

The Gastroenterological Association of Thailand

Tel: 662 256 4265 Fax: 662 253 8272

E-mail: fmedvmc@md2.md.chula.ac.th

11th Asian Pacific Congress of Gastroenterology & 8th Asian Pacific Congress of Digestive Endoscopy

Date : March 10-14, 2000

Place: Hong Kong Convention & Exhibition Centre

For further information

Veronica Cheng, Orient Dynamic Company Limited.

Tel: 2736 7837 Fax: 2376 0329 E-mail: onh@netvigator.com

Joint HKSGE, HKSDE and HKSCP Scientific Meeting Scientific Program

13:30 - 13:55	Registration		
13:55 - 14:00	Opening Remark		JY Sung
14:00 - 15:15	Session One	GERD Chairmen WM Hui, JY Sung Epidemiology Motility, pH and H. pylori Medical vs Surgical Rx	W Hu H Yuen L Lundell
15:15 - 16:30	Session Two	Colo-rectal Cancer Chairmen F Cheng, ML Szeto Screening Endoscopy Surgery	S Kwok F Mok M Li
16:30 - 17:00	Coffee Break		
17:00 - 18:00	Session Three	Free Paper Presentation Chairmen KM Lam, YW Luk	
18:00 - 19:30	Dinner Symposium	Gastrointestinal Bleeding Chairmen S Chung, SK Lam H. pylori NSAID Acid Suppression Endoscopic Treatment & Re-treatment Lower GI Bleeding	KC Lai KL Chan L Lundell J Lau WY Lau
19:30 - 20:00	Cocktail		
20:00 - 22:00	Dinner	Prize Presentation	

Venue : Grand Ballroom, Lower Level 1, Kowloon Shangri-la Hotel, 64 Mody Road, Kowloon

For Registration, please contact Miss Winnie Lam at 2420 7388

C.M.E.

ACCREDITATION

The College of Surgeons of Hong Kong

The Hong Kong College of Physicians

The Hong Kong College of Family Physicians

The Hong Kong College of Pathologists

The Hong Kong College of Radiologists

(5.5 CME Points)

(3.5 CME Points)

(4 CME points)

(5 CME points)

(4 CME points)

Scholarship

Objective

The objective of the Scholarship is to facilitate trainees in gastroenterology (physicians, surgeons, radiologist, pathologists) of the Hospital Authority or the Society to undertake overseas training in gastroenterology.

Award

1. The Scholarship is awarded for a study period of 6 months or more.
2. The award is HK\$50,000.
3. Simultaneous acceptance of other scholarship is not allowed.
4. The candidate is required to produce evidence of approval to take study leave from his/her serving institution.
5. Upon completion of the training programme, the candidate is required to submit a report to the Society within 2 months, together with original letter of approval on study leave.
6. The award will be paid on satisfactory completion of the training programme
7. An interview may be required by the Scholarship Selection Committee
8. The Scholarship Selection Committee has the sole discretion to grant the Scholarship or to refuse any application.

Qualifications

Application is limited to fellow trainees in Gastroenterology who are:-

1. staff of Hospital Authority or members of the Society
2. holder of a post-graduate diploma (MHKCP/MRCP(UK) or equivalent)
3. in the Hong Kong Academy of Medicine Fellowship Training

Application

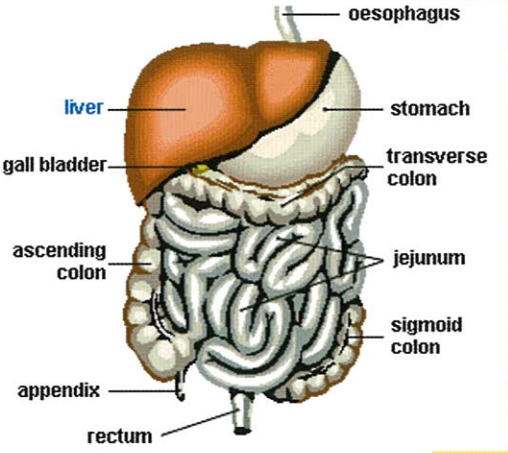
1. Interested parties should apply in writing enclosing the curriculum vitae (which should include personal data, professional qualifications, working experience, training experience in gastroenterology, publications and proposed training programme, as well as the reference from at least one supervisor or a referee if the applicant is in private practice)
2. Application should be sent together with supporting documents to the Scholarship Selection Committee, Hong Kong Society of Gastroenterology c/o Miss Clare Wong, Department of Medicine, Queen Mary Hospital, Pokfulam Road, Hong Kong (Tel: 2855-3354, Fax: 2816-2863 and E-mail: mcwong@hkucc.hku.hk)
3. Deadline of application is on April 30, 1999

Where are we ?



HONG KONG SOCIETY OF GASTROENTEROLOGY

- Objectives
- Background
- Office Bearers & Past Presidents
- Membership
- Events & News
- Abstracts
- Links



The Society has its own Website at <http://medicine.org.hk/hksg/home.htm>