Hong Kong Society of Gastroenterology

JUNE 2003

Message from Professor Joseph Sung, President

The past two months have been a very challenging and stressful time for everyone living in Hong Kong, in particular our medical professionals and healthcare practitioners. Severe acute respiratory syndrome (SARS) has been creating such an havoc in clinics and hospitals as well as in the community. Day and night, we are fighting to combat this disease and pray that the epidemic will soon over.

The impact of the SARS on our gastroenterological circle was equally pounding. Two of our scientific meetings scheduled to be held in Hong Kong in April and May have been cancelled or deferred. Two major international conference: EASL and APDW were deferred.

On the other hand, we are seeing great opportunities amidst this disaster. New medical innovations, drugs and treatments emerge through the coordinated efforts of local and world medical experts. From a wider perspective, we believe that SARS will change the practice of Medicine, and indeed, the life of many individuals.

The Hong Kong Society of Gastroenterology is fortunate to be able to hold its Annual General Meeting and Scientific Meeting on March 20, 2003 as scheduled. I apologize for not being able to chair the meeting on that day, lest I would infect the GI community in Hong Kong. I was given to understand the meeting went very well with a high level of interactive discussions. Majority of the participants stayed through the meeting and enjoyed the dinner. I would like to pay my tribute to Dr. M L Szeto for conducting the meeting in my absence and Dr. Judy Ho for organizing such an excellent scientific session.

This issue of the Newsletter contains valuable topical issues on *Pathology perspective of "non-specific colitis"* by Dr. S T Yuen and *Cancer Marker* by Drs. Wilson Tsui, William Lo and Francis Mok. I wish to thank the contributors for their remarkable research and scientific updates in the two specialized areas. I am sure our readers will find these articles useful and enlightening.

Last but not the least, may I express my heartfelt gratitude to many colleagues and friends who send us their blessing, cards, flowers, fruits, emails... during this very difficult time. Solidarity and perseverance will give us final success in this battle. Let us build Hong Kong again with our heart and our blood. May I wish all of you good health and happiness!

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Scientific Updates

Pathology perspective of "non-specific colitis" - What to do?

Dr. Yuen Siu Tsan
Department of Pathology
Queen Mary Hospital

It is well known that the colorectal mucosa has limited ways to respond to injuries and stresses, and the similarities and overlap of pathological features in ulcerative colitis, Crohn's disease and other colorectal inflammatory responses causes considerable diagnostic confusion. On the other hand, there may be a lack of awareness of the range of normal colorectal histology and the minimal features that indicate clinically important inflammation on the side of the pathologists. Moreover, effective clinical management depends a lot on the accurate pathological diagnosis, and thus it is important for an "interpretative pathological diagnosis" to be clearly conveyed to the clinicians in-charge of the patient. Many a times, a correct pathological diagnosis can only be made when information regarding the clinical and endoscopic findings, unfortunately not adequate in most of the requesting forms, is integrated. At times, when a specific diagnosis cannot be reached, a diagnosis of "non-specific colitis" is issued. This diagnosis is confusing in helping the clinician to make clinical decisions and sometimes reflect the pathologist's inability to judge the limits of normal range (1 - 3). To deal with this problem, a set of guidelines and structured approach has been developed to help pathologist in colorectal biopsy assessment (4, 5). Here, I would like to highlight some of the essential points. For details, the readers should refer to the two original publications.

When assessing colorectal biopsy specimens, a pathologist should ask oneself of the following sequence of questions based on evidence-based guidelines (4, 5):

1. Is the mucosa normal or inflamed?
2. Are there features of chronic inflammatory bowel disease?
3. If the features suggest chronic inflammatory bowel disease, do they support a diagnosis of ulcerative colitis or Crohn's disease?
4. Are there features suggestive of

acute infective-type colitis?
5. Do the features represent another form of inflammation?

1. Is the mucosa normal or inflamed?

Pathological changes are usually assessed in the following categories: mucosal architecture, mucosal inflammation/cellularity, polymorphs infiltration and epithelial abnormality. A normal mucosa is one with normal crypt density, undistorted crypt architecture, flat mucosal surface, normal density and distribution of lamina propria cells, no excessive neutrophil infiltration, intact normal surface epithelium and normal mucin content of goblet cells. However, there are ranges of appearances of "normal" colorectal mucosa and it is important that the pathologist knows and can therefore accurately classify those as "normal". The composition and distribution of the lamina propria cellularity is one of the important feature with which a pathologist would classify cases as having "inflammation" or not. It should be noted that in the normal large bowel, most cells are situated in the upper third of the lamina propria and the ratio of cellularity between the superficial and the basal thirds is about 2:1 (1, 2). A mild diffuse increase in the superficial portion of the lamina propria should be interpreted with caution and should only be diagnosed in the absence of other pathological changes IF the pathologist is confident that it is NOT normal. Many a times, a diagnosis of "mild non-specific inflammation" is entered just because there is a "mild" increase of cellularity in the upper lamina propria, which may be "normal". The recognition of the usual presence of lymphoid follicles is also important. Lymphoid follicles can be found in normal mucosa and are often of full mucosal thickness. These can extend into submucosa and may even incorporate crypts to form "lymphoglandular complexes" (6, 7). Vigorous bowel preparation may result in the presence of 1 or 2 neutrophils in an occasional crypt and surface epithelium (2). It should be noted that the normal distal rectum may show crypt shortening with increase spacing and branching. The anorectal junction may show features similar to mucosal prolapse, and therefore should be avoided for diagnostic biopsy (8). Finally, if there is doubt about the normality of the biopsy, multiple levels should be examined.

2. Are there features of chronic inflammatory bowel disease?

If the colonic mucosa is inflamed, then one should decide if it is chronic inflammatory bowel disease or infective colitis. Surface irregularity, decreased crypt density and crypt architectural distortion are features that suggest chronic inflammatory bowel disease. A moderate or severe transmucosal increase of chronic inflammatory cells in the lamina propria also suggests CIBD. Inflammatory infiltrated is more in the superficial lamina propria in infective colitis. It should be noted that the severity of inflammatory infiltration depends on the disease activity at the time of biopsy. True epithelioid cell granulomas strongly suggest Crohn's disease but mucin containing histiocytes can be present in the normal mucosa and histiocytic reaction towards disrupted crypt should not be confused with granuloma. There is a wide overlap in neutrophil infiltration between infective colitis and CIBD depending on timing of biopsy in relation to disease onset and activity.

3. If the features suggest chronic inflammatory bowel disease, do they support a diagnosis of ulcerative colitis or Crohn's disease?

The features that suggest ulcerative colitis are severe crypt architectural distortion, severe widespread decreased crypt density, villous surface, diffuse heavy transmucosal lamina propria cell increase and severe mucin depletion. Features to suggest Crohn's disease are epithelioid granulomas, discontinuous crypt distortion and inflammation, and focal cryptitis. It should be noted that histological features alone are not sufficient in making the final definitive diagnosis, which usually depends on assessment of the clinical history, radiological and endoscopic findings, examination of sequential biopsies and biopsies from multiple sites.

4. Are there features suggestive of acute infective-type colitis?

If inflammation is present but the features are not diagnostic of CIBD, then infective colitis and other etiologies have to be considered. It should be noted that the features that are associated with infective colitis are seen only in the early acute phase. Many a times, patients are investigated after several weeks or even months after the onset of symptoms. The increase in lamina propria cellularity and neutrophil infiltration including cryptitis and crypt abscess, is usually present in the superficial portion of the mucosa. It should also be noted that the features of CIBD and infective colitis may overlap and that only the follow up clinical course and histological changes will tell the final diagnosis.

5. Do the features represent another form of inflammation?

Features of specific diseases such as microscopic colitis, ischaemic colitis, graft-versus-host disease should be looked for. Finally, reappraisal of the clinical and histopathological features, with follow up of the clinical course and repeat biopsies from multiple sites are needed for a definitive diagnosis.

As can be seen from the above, a diagnosis of "non-specific colitis" can be given in the following settings:

a. The biopsy may actually be in the upper range of normal, which the pathologist is somewhat uncomfortable to just call it "normal".

b. There is definite "inflammation" but the histological features are not typical and that the insufficient clinical information given makes it not possible for the pathologist to make a confident interpretative stratification.

c. There is definite "inflammation" and even with the adequate information, it is not possible to stratify the histological changes into a specific

disease category. This category should be better termed "inflammation - unclassified".

What to do then?

The most important thing to do is to discuss with the pathologist and provide in more detail information regarding the clinical history and endoscopic findings. The pathologist may review the colorectal biopsy. The additional information may help the pathologist to stratify the case into specific disease category. For those that fall into the category "inflammation-unclassified", further investigation and follow up of the patient with repeat biopsies from multiple sites along the large bowel may reveal the final diagnosis.

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CANCER MARKERS : CLINICAL SIGNIFICANCE - CEA, CA19.9, CA72.4

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Introduction

Cancer is among the leading causes of death in developed countries, and it is estimated that a third of all people in western Europe develop a malignant disease at least once in their lifetime. Important in the management of cancers, serum tumour markers have been developed for diagnosis, population screening, staging, prognostication, monitoring treatment, detecting recurrence, and various purposes. A tumour marker, by definition, is a substance present in or produced by a tumour or by the tumour's host in response to the tumour's presence that can be used to differentiate a tumour from normal tissue or to determine the presence of a tumour based on measurement in blood or secretions. In the history of tumour markers, the discovery of oncofetal antigens in 1960's have led to a general application of these markers for monitoring cancer patients. With the development of monoclonal antibodies in 1970's, new antigens derived from tumour cell lines, such as various carbohydrate antigens, were discovered. In the last two decades, the advances in molecular genetics have led to rapid understanding and use of tumour markers at the molecular level (1,2). For the cancers of gastrointestinal tract, carcinoembryonic antigen (CEA), carbohydrate antigen determinants CA19.9, and the more recent, CA72.4 are useful markers, although they are also expressed in cancers of other organs, including breast, lung, and ovary. In particular, they complement each other and play an important role in the management of patients with gastrointestinal malignancies.

Carcinoembryonic Antigen Biochemistry

CEA, first discovered by Gold and Freeman and currently the most clinically useful oncofetal marker, is a high-molecular-weight glycoprotein present in colonic adenocarcinoma and fetal gut (3). Evidence suggests that CEA can act as a cellular adhesion molecule that may potentiate invasion and metastasis (4). The CEA family consists of about 10 genes located on chromosome 19. The serum CEA is detected by immunoassay and the immunodeterminant portion is found on the protein portion of the molecule.

Clinical application

In the healthy population, the upper limit of CEA is about 3 ng/ml for non-smokers and 5 ng/ml for smokers. Serum CEA is elevated in colorectal carcinoma (70%) as well as a variety

of cancers such as gastric (50%), pancreatic (55%), lung (45%), breast (40%), uterine (40%) and ovarian (25%). It is relatively insensitive in early stages of malignant disease, because CEA level reflects tumour bulk. It is also elevated in some patients having benign conditions such as cirrhosis (45%), pulmonary emphysema (30%), rectal polyps (5%), benign breast disease (15%), and ulcerative colitis (15%). Because of the elevations associated with benign diseases (i.e. false-positive results) and the number of tumours that do not produce CEA (i.e. false-negative results), CEA testing is not a useful screening or diagnostic tool (5).

Although CEA assays have not been as useful as had been envisaged initially in the screening and diagnosis of colonic carcinoma, they are important as an adjunct to clinical staging and as a prognostic indicator (6). Preoperative serum CEA levels have been found to correlate inversely with tumour grade and directly with pathological stage. It is elevated in 28% of patients with Duke's stage A colorectal cancer, 45% of those with stage B and 70% of those with stage C. The pretreatment CEA level is also prognostic of the development of metastasis. A high level of CEA is associated with a greater likelihood of developing metastasis. CEA concentration of <5 ng/ml before therapy suggests localized disease and a favourable prognosis, but a concentration of >10 ng/ml suggests extensive disease and a poor prognosis; >80% of colon carcinoma patients with values >20 ng/ml have recurrence within 14 months after surgery. Increased values in node-negative colon cancer may identify poorer-risk patients who may benefit from chemotherapy.

At the present time, treatment monitoring of colorectal cancer is the most useful area for CEA testing. After successful initial treatment, CEA levels decline to the normal range in 6-12 weeks. During remission, CEA levels are stable. If the CEA levels fail to fall to the normal range after surgery, it is likely that the resection has been incomplete or that the cancer had already metastasized. A sustained and progressive postoperative CEA rise signifies tumour recurrence. In a study of 300 colorectal cancer patients followed post-operatively with serial serum CEA, the observation of two consecutive elevations in the CEA level yielded a sensitivity of 84% and a specificity of 100% in the detection of recurrence, and in 72% of cases preceded all other clinical signs (7). The lead time from CEA elevation to clinical recurrence is about 5 months. Although the best evidence for this phenomenon is found in colorectal cancer, similar data have been obtained in other CEA-expressing gastric, pancreatic, breast and lung carcinomas. The newer application is the use of labelled anti-CEA antibodies to localize colonic tumours in imaging and to target therapy directly to cancer cells.

CA19.9

Biochemistry

CA19.9 is a carbohydrate antigen occurring as a glycolipid in tissues and as a mucin-type glycoprotein in serum. It is synthesized by normal human pancreatic and biliary ductular cells and by gastric, colonic, endometrial, and salivary epithelia. This carbohydrate antigen is a sialylated derivative of the Lewis^{a} blood group antigen and its expression requires a Lewis gene product. Patients who are genotypically Lewis$^{a\text{-}b\text{-}}$ (about 5%) do not express CA19-9, a fact implying that the maximal achievable sensitivity of the marker will not be > 95%. The monoclonal antibody against CA19.9 was developed from a human colon carcinoma cell line, SW-1116, by Koprowski and co-workers (8). It is detected by immunoassay and the immunodeterminant structure resides on the carbohydrate side chains of the molecules.

Clinical application

CA19.9 is a marker for both pancreatic and colorectal carcinoma (9). The upper reference limit is 37 U/ml. Elevated levels were seen in patients with pancreatic (80%), hepatobiliary (67%), gastric (40-50%), hepatocellular (30-50%), colorectal (30%), and breast (15%) cancers. Pancreatitis, cholangitis, cirrhosis and other benign gastrointestinal diseases show a 10 to 20% elevation; however the levels are usually lower than 120 U/ml.

For pancreatic carcinoma, CA19.9 is the most useful blood test in the diagnosis and management (10). The good sensitivity (79%) and high specificity (90%) have made it a superior marker over CEA for pancreatic cancer. If higher cutoffs are used, the specificity rises so that, at levels >1000 U/ml, the marker's specificity approaches 100%. It may also be used to indicate development of cholangiocarcinoma in patients with primary sclerosing cholangitis.

CA19-9 levels also correlate with pancreatic cancer stage, and high preoperative levels are related to a poor prognosis. With the cut-off of 37 U/ml, 67% of patients with resectable and 87% of those with unresectable pancreatic cancer show elevated values. By raising the cut-off, 96% of the tumours with blood levels >1000 U/ml have been found to be unresectable. CA19-9 is useful in monitoring pancreatic and colorectal cancer as well. After potentially curative surgery, patients who normalize their CA19-9 postoperatively live longer than those who do not. Furthermore, serial assays can indicate recurrence prior to radiographic or clinical findings by 1 to 7 months. Hence, CA19.9 is an excellent guide to diagnosis and response to treatment for patients with pancreatic cancer.

CA72.4

Biochemistry

CA72.4 detects the tumour-associated glycoprotein TAG-72 isolated from breast carcinoma and colon carcinoma cell lines, being identified by monoclonal antibodies B72.3 and cc49 respectively. It is detected by a double determinant immunoassay employing both antibodies.

Clinical application

CA72.4 is a marker for carcinomas of the gastrointestinal tract and of the ovary (11). A cut-off of 4 U/ml is used in the assay. The following percentages of elevation were observed: healthy subjects, 3.5%; benign gastrointestinal diseases, 6. 7%; gastrointestinal carcinoma, 40%; lung cancer, 36%; and ovarian cancer, 24%. Compared with the previous two markers, CA72.4 appears to have a higher tumour specificity (98%), so that elevated serum levels of CA72.4 should always be taken seriously (12).

Tumour-indicating sensitivity of CA72.4 is clearly inferior to that of CA19.9 in pancreatic carcinomas (22% Vs 82%; all stages) and to that of CEA in colorectal cancer (32% Vs 58%; all stages). However, in gastric carcinoma CA72.4 identified 59% of all patients (CA19.9, 52%; CEA, 25%), and a combination of CA72.4 and CA19.9 detected as many as 70% (12). Positive results correlated roughly with tumour size. It is a useful marker of late stage gastric carcinoma (13).

The combined assay of CA72.4, CA19.9 and CEA provides prognostic information in patients resected for gastric cancer. Patients with preoperative positivity for one of these three markers should be considered at high risk of recurrence even in early stages of gastric carcinoma (14). A greater risk of death is found with high preoperative serum CA72.4 level (15). Serial combined assays are also useful for in the follow-up and early diagnosis of recurrence, achieving a combined sensitivity of 100% in patients with positive preoperative levels. However, only CA72.4 positivity (specificity 97%) should be considered a specific predictor of tumour recurrence, while CEA and CA19.9 frequently yielded false positives (16).

Conclusion

An ideal tumour marker should be both specific for a given type of cancer and sensitive enough to detect small tumours for early diagnosis or during screening.

Table : Clinical use of CEA, CA19.9 and CA72.4 in gastrointestinal malignancies

Tumour site	Screening	Detection & diagnosis	Staging & prognosis	Follow - up
Colorectal			(CEA)	CEA
Gastric#			CA72.4	CA72.4
Panocreatic		CA19.9	CA19.9	CA19.9

CA72.4 is not sensitive enough and combined assay of all 3 markets recommended.

Unfortunately, most tumour markers known today are neither specific nor sufficiently sensitive for such purposes. Tumour markers are most useful in evaluating the progression of disease status after the initial therapy and in the monitoring of subsequent treatment modalities. A complementary role is established for CEA, CA19.9 and CA72.4 (Table) in the management of gastrointestinal cancers, although the clinical utility is somewhat limited by the false positivity, false negativity, and relatively ineffective treatment protocols for recurrent cancers.

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Highlights from The Annual General Meeting and Scientific Meeting 2003

Dr. Judy Ho, Senior Medical Officer, Queen Mary Hospital

March 20, 2003 (Thursday)
Ballroom, 3/F Sheraton Hotel & Towers
6:15 - 10:00 p.m.

Scientific Meeting

The Interactive Digestive Disease Case Discussion formed a major of the evening's programme. The meeting started at 7:10 p.m. with an introduction by Dr. Judy Ho, the Organizing Chairman. There were altogether three case presentations as follows :

Case 1: *A man with chronic bloody diarrhea* by Dr. Leong In Son of Pamela Youde Nethersole Eastern Hospital

Case 2: *A young man with diarrhoea and pneumaturia*, a Queen Mary Hospital case by Dr. Hui Aric Josun on behalf of Dr. Raymond Wong who apologized on account of sickness

Case 3: *A lady with sudden onset of epigastric pain and jaundice* by Dr. Law Siu Tong of United Christian Hospital.

The 3 panel chairmen were Dr. Szeto Ming Leung, Dr. Lai Moon Sing and Dr. Lai Jak Yiu and the panel discussants for Cases 1 to 3 respectively included:

Drs. Li Kam Fu and Lok Ka Ho of Tuen Mun Hospital

Dr. Yiu Chi Him of Alice Ho Miu Ling Nethersole Hospital (Dr. A J Hui assumed the role of Case 2 presenter) and,

Drs. Ambrose Kwan and Wong Wing Hang of Princess Margaret Hospital.

The meeting went well. About 180 doctors attended the scientific session. There were active discussions following each presentation.

Seven exhibition booths by the biomedical industry in Hong Kong were set up. Participants paid visits to the exhibition booth and talked to the company representatives both at registration time and during the Annual General Meeting. The AGM was also smoothly conducted. 44 members and fellows were present. A few important resolutions were passed. Majority of the participants joined the after-meeting dinner. In token of appreciation of the contributions from concerned parties: invited panel chairman, panel discussants, case presenters and pharmaceutical companies, this Society presented at dinner to each a souvenir in the form of a Society plaque.

This Society wishes to add its thanks to the following companies for their contributions and sponsorship: Abbott Laboratories, Altana Pharma, AstraZeneca, Eisai, Novartis, Roche and Takeda.

Fifth Joint Annual Scientific Meeting

Venue : Miramar Hotel

Date & Time : September 27, 2003
1:00 - 9:00 p.m.

Co - organizers : HK Society of Gastroenterology
HK Society of Digestive Endoscopy
HK Society for Coloproctology
HK Association for the Study of Liver Diseases
HK Society of Gastrointestinal Motility

Sessions and Topics :

Free Paper

Upper Gastrointestinal Bleeding
- Preventing GI Toxicity of NSAIDs
- IV PPI in the management of bleeding ulcers

Jaundice
- New Treatments for HBV Infection
- New treatments for obstructive jaundice

Abdominal Pain
- Pancreatitis : Imaging & intervention
- Irritable bowel syndrome in Hong Kong

Sponsors : AstraZeneca GlaxoSmithKline

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News ...

Sponsorship to attend international conferences

With immediate effect, sponsorship to attend an international conference will be placed on the HKSGE web for general notice for 14 days as soon as this is received from the relevant sponsor. In exceptional cases when a sponsorship is received at short notice, posting on web will be less than the normal 14 days*. Interested members and fellows are hereby advised to send in their application for consideration before the specified closing date.

The Council of this Society will assess applications based on the following criteria and decide on the award. Its decision shall be final.

Assessment criteria :

- membership : applicant must be a paid-up member/fellow of this Society for at least a year by the closing date of application
- appropriateness of the meeting to the applicant
- role played by the applicant in the meeting such as speaker, session chairman, abstract presenter etc.
- applicant's contributions to this Society, Hong Kong or internationally in the specialized field
- any other contributing factors as may be relevant.

All sponsorship payments should be addressed to **"The Hong Kong Society of Gastroenterology Ltd."** which will reimburse the successful applicants as appropriate on production of original receipts. No payment or any part thereof shall be made by the sponsor direct to the applicant.

** Please allow at least 2 working days for the web hosting company to put up the sponsorship information.*

HKSGE web

This Society has now its own web *www.hksge.org* which contains updated information on its progress and development and special features such as the Newsletter Section, Secretariat and Message Board with a contact email address *gastro@hksge.org.* Members and fellows are welcome to pay frequent visits to the web and provide their views on Society matters and professional advancement.

www.hksge.org

Major Meetings

May 30-June 1, 2003
Australian Liver Association Hepatology Research Workshop
Organizer: Australian Liver Association
Location: Crowne Plaza Hotel, Coogee Beach, Sydney
Website: www.gesa.org.au

June 5-7, 2003
Prague Hepatology Meeting
Organizer: The Czech Society of Hepatology
Location: Hotel Hilton Prague, Czech Republic
Website: www.czech-hepatology.cz

June 18-21, 2003
9th Congress of the International Liver Transplantation Society
13th Congress of the Liver Intensive Care Group of Europe
Organizer: International Liver Transplantation Society
Location: Barcelona (Spain)
Website: www.oasismeetings.com

July 9-12, 2003
The Indonesian Digestive Disease Week
Organizer: Indonesian Society of Gastroenterology
Location: Discovery Kartika Plaza/Hotel Kuta Bali

For further information, please contact -
Tel: 62 2131 53957 Fax: 62 21 31 42454

August 7-10, 2003
The South African Digestive Disease Week 2003
Organizers: South African Gastroenterology Society, African mid-East Association of Gastroenterology, World Organisation of Gastroenterology, South African Society of Endoscopy Surgeons, South African Society of Enteral and Parenteral, Nutrition and South African Gastrointestinal Nurses Society
Location: Sandton Convention Centre, Johannesburg, South Africa
Website: www.saddw.co.za

August 28-31, 2003
Seventh Congress of Asian Society of Hepato-Biliary Pancreatic Surgery
Organizer: Asian Society of Hepato-Biliary Pancreatic Surgery
Location: Sri Ramachandra Medical Ainstitute, Porur Chennai, India
Website: www.ashbps2003.com

September 27, 2003
5th Joint Annual Scientific Meeting
(see Programme on P.6)

Welcome !!!

New Fellow

Dr. Lao Wai Cheung

Department of Medicine
Pamela Youde Nethersole Eastern Hospital

New Member

Dr. Wong Wing Hang

Department of Medicine
Princess Margaret Hospital

October 7-10, 2003
Australian Gastroenterology Week 2003
Organizer: Gastroenterological Society of Australia
Location: Cairns Convention Centre, Cairns Queensland
Website: www.gesa.org.au

2003年10月17-18日
中華醫學會消化病學分會
2003年胃腸動力會議
組織者：中華醫學會消化病學分會
地點：天津
網址：www.csge.org

November 19-21, 2003
10th Hong Kong International Cancer Congress
Organizer: The Faculty of Medicine of The University of Hong Kong and The Cancer Centre of Queen Mary Hospital
Location: to be announced
Website: www.hkicc.org

November 28-30, 2003
10th Anniversary Celebration Programme "New Challenges in Healthcare"
Organizer: The Hong Kong Academy of Medicine
Location: HKAM Jockey Club Building, 99 Wong Chuk Hang Road, Aberdeen, Hong Kong
Website: www.hkam.org.hk

Asian Pacific Digestive Week 2003
Singapore, July/August 2003
Raffles City Convention Centre, Singapore
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Madrid, Spain, November 1-5, 2003
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