SOUVENIR PROGRAMME & ABSTRACT BOOK

ANNUAL SCIENTIFIC CONGRESS OF THE
MALAYSIAN SOCIETY OF GASTROENTEROLOGY AND HEPATOLOGY

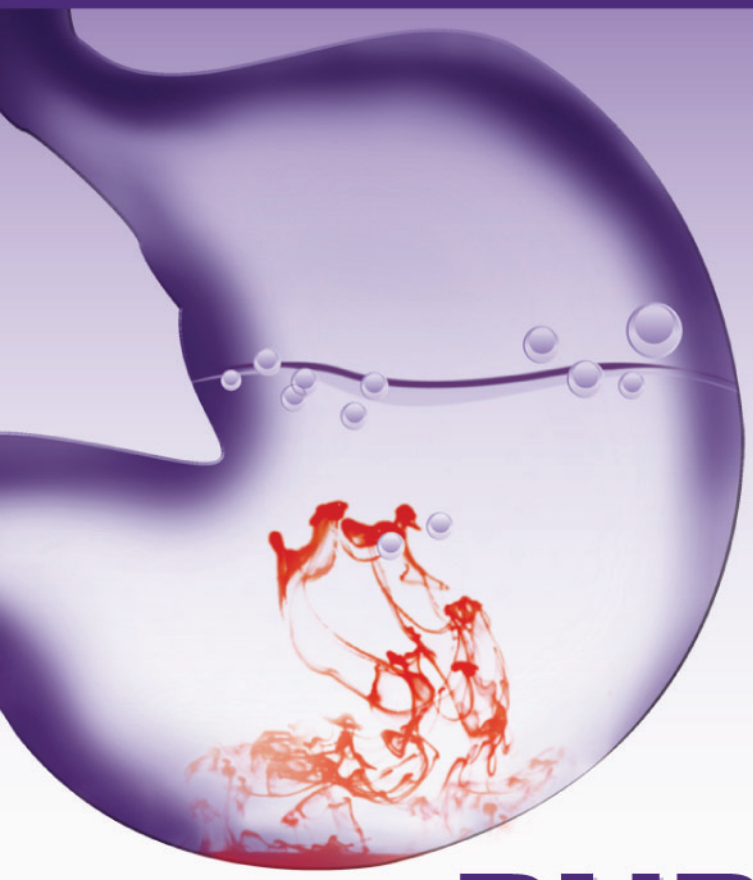
GUT 2016

22nd to 24th July 2016

Shangri-La Hotel
KUALA LUMPUR, MALAYSIA

The PPI approved for Prevention of Peptic Ulcer re-Bleeding[1]

Nexium® esomeprazole



- Significant reduction of re-bleeding within 3 days, sustained up to 30 days[2]
- Achieves pH>4 faster than pantoprazole iv[3]
- Maintains pH>6 for 12.6 hours [4]

PUB 30

3 days[2] + 27 days[2]

80 mg iv bolus
8 mg/h iv infusion

40 mg
MUPS tablets

References
1. Nexium® Prescribing Information. 2. Sung JJY et al. Ann Intern Med 2009;150(7): 455-64. 3. Clive H. Wilder Smith et al. Alimentary Pharmacology & Therapeutics 2004, 20:1099-104. 4. K Rohss et al. Int. J of Clin. Pharma & Therapeu, Vol 45-No5/2007 (345-354)

Abbreviated prescribing Information:

Nexium® (Esomeprazole). Film-coated tab (MUPS) 20 mg x 14's. 40 mg x 14's. **Indications:** listed in dosage. **Dosage:** *Adults and adolescents from the age of 12:* Treatment of erosive reflux oesophagitis: 40 mg once daily for 4-8 weeks. Long-term management of patients with healed oesophagitis to prevent relapse: 20 mg once daily. Symptomatic treatment of GERD: 20mg once daily in patients without oesophagitis until symptom control is achieved. If control has not been achieved after 4 weeks, the patient should be further investigated. Once symptoms have resolved, subsequent symptom control can be achieved using 20 mg once daily. In adults, an on demand regimen taking 20 mg once daily, when needed, can be used. On demand regimen not recommended in NSAID treated patient at risk of gastric cancer and duodenal ulcer. Eradication of *H. pylori*, healing of *H. pylori* associated duodenal ulcer and prevention of relapse of peptic ulcers in patients with *H. Pylori* associated ulcers: 20 mg Nexium with 1 g amoxicillin and 500 mg clarithromycin, all b.i.d for 7 days. Healing of gastric ulcers associated with NSAID therapy: 20 mg once daily for 4-8 weeks. Prevention of gastric and duodenal ulcers associated with NSAID therapy in patients at risk: 20 mg once daily. Prolonged treatment after I.V induced prevention of rebleeding of peptic ulcers: 40 mg once daily for 4 weeks after IV induced prevention of rebleeding of peptic ulcers. Treatment of Zollinger Ellison Syndrome: 40 mg b.i.d. The dosage should then be individually adjusted and treatment continued as long as clinically indicated. Majority of patients can be controlled on doses between 80 mg to 160 mg daily. With doses above 80 mg daily, the dose should be divided and given b.i.d. **Contraindications:** Known hypersensitivity to esomeprazole, substituted benzimidazoles or any other constituents of the formulation, nelfinavir. **Precautions:** Exclude gastric malignancy prior to treatment. Fructose intolerance, glucose-galactose malabsorption or sucrase-isomaltase insufficiency. Co-administration of esomeprazole with atazanavir is not recommended. If unavoidable, close clinical monitoring is recommended in combination with an increase in the dose of atazanavir to 400 mg with 100 mg of ritonavir; esomeprazole 20 mg should not be exceeded. **Undesirable effects:** Headache, abdominal pain, constipation, diarrhoea, flatulence, nausea/vomiting.

Nexium® (Esomeprazole) 40 mg Injection/Infusion. **Indications:** When oral route is not possible or appropriate; treatment of gastroesophageal reflux disease in patients with esophagitis and/or severe symptoms of reflux, healing of gastric ulcer associated with NSAID therapy and prevention of gastric and duodenal ulcer associated with NSAID therapy. Prevention of rebleeding following therapeutic endoscopy for acute bleeding gastric or duodenal ulcers. **Dosage:** Reflux oesophagitis: 40 mg once daily. Reflux disease (symptomatic treatment): 20mg once daily. Healing of gastric ulcer and prevention of gastric and duodenal ulcer associated with NSAID therapy: 20mg once daily. Treatment with Nexium IV can be given for up to 10 days as part of a full treatment period for the specified indications. Prevention of rebleeding of gastric and duodenal ulcers: following therapeutic endoscopy for acute bleeding gastric or duodenal ulcers, 80 mg should be administered as a bolus infusion over 30 minutes, followed by a continuous intravenous infusion of 8 mg/h given over 3 days (72 hours). The parenteral treatment period should be followed by oral acid suppression therapy. **Contraindications:** Hypersensitivity to the active substance esomeprazole or to other substituted benzimidazoles or to any of the excipients, nelfinavir. **Precautions:** Exclude gastric malignancy prior to treatment. Co-administration of esomeprazole with atazanavir is not recommended. If unavoidable, close clinical monitoring is recommended in combination with an increase in the dose of atazanavir to 400 mg with 100 mg of ritonavir; esomeprazole 20 mg should not be exceeded. **Undesirable effects:** Headache, abdominal pain, constipation, diarrhea, flatulence, nausea/vomiting.

Further information available on request
Please consult local full prescribing information before prescribing
For Healthcare Professional Only

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HON TREASURER	Dr Tee Hoi Poh
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COMMITTEE MEMBERS	Dr Hamizah Razlan Datuk Dr Raman Muthukaruppan Dr Ramesh Gurunathan Prof Dr Sanjiv Mahadeva Dr Tan Soek Siam Dr Tee Hoi Poh

MESSAGE FROM THE PRESIDENT, MALAYSIAN SOCIETY OF GASTROENTEROLOGY AND HEPATOLOGY & ORGANISING CHAIRPERSON, GUT 2016

The Organising Committee of GUT 2016 would like to extend a warm welcome to all delegates, speakers, and sponsors. This conference has been drawn up to keep the participants updated in the current trends in gastroenterology, hepatology and GI surgery. The programmes has been fine-tuned to meet the expectations of allied health professionals, trainees in medical and surgical post-graduate programmes, the General Physician, the General Surgeon, as well as the Specialist Gastroenterologist, Hepatologist and Surgeon.

The Organising Committee has put together an attractive scientific programme which includes plenary sessions, lunch and tea symposia, video sessions and case discussions. The entire meeting has been centered on both the basics, as well as bringing you the very latest in the field of Gastroenterology and GI Surgery. We have an impressive list of local and world-renowned speakers invited for the conference, with a focus on practical points to take home. We have had a great response from the trainees with their research, and there will be both oral and poster presentations.

Over the years, this meeting has enabled many to meet up with old friends and colleagues, as well as spending time with the family. The social programme through the hotel, provides an interesting variety for both the delegates, as well as the accompanying persons.

Finally, we would like to thank all the invited speakers, delegates and sponsors for their commitment and time to ensure a successful meeting for all. I would hope that you will find this conference rewarding, both academically, as well as socially. I hope that you will have an enjoyable stay and I look forward to meeting you at the conference.

Dr Mohamed Akhtar Qureshi

16TH MSGH DISTINGUISHED ORATOR – PROFESSOR DR KENNETH E L McCOLL

Citation by Professor Dr Lee Yeong Yeh

Helicobacter pylori and the Pathophysiology of Gastroduodenal and Oesophageal Disease

Kenneth E L McColl, known as Ken to many, is Professor of Gastroenterology in his alma mater, University of Glasgow, since 1992. He is much regarded as a par-excellence clinical researcher in his homeland of Scotland but also beyond, and he is a much sought-after research supervisor by many gastroenterology trainees in the UK. I have often heard from his peers that he is one of the best researchers in the field, and I must say I am very proud that he is my mentor. He is no stranger to many in Malaysia. Since his first talk back in 1998, in the Helicobacter Congress held in Kota Kinabalu, he has supported us time and again, in subsequent GUT meetings in 2005 and 2013, as well as the APDW in 2010. On the other hand, Malaysia is no stranger to Professor McColl too. He fondly remembers his younger days being an adventurous 20-year old medical student. He had spent three months in Asia with stops including Kuala Lumpur but also a Mission Hospital in the very South East corner of Bangkok – just across the border from Kota Bharu!

Being a brilliant student, he obtained his medical degree with commendation from the University of Glasgow in 1974, and he subsequently undertook training in general internal medicine and gastroenterology. Being trained by a caring but yet highly competent physician, Sir Thomas J Thomson, Professor McColl is an equal match to his clinical teacher. In 1980, he was awarded a Medical Research Council Travelling Fellowship where he spent a year in the University of California, San Francisco. He has been appointed as Consultant Physician in Gastroenterology in the Western Infirmary, Glasgow, since 1984, but he also serves at the Gartnavel Hospital until today. The Dyspepsia Research Clinic in this hospital was the place where I spent most time during my PhD with Professor McColl.

The early research career of Professor McColl was greatly influenced by the porphyria work of Sir Abraham Goldberg. He was awarded a MD with Honours and Bellahouston Medal in 1981 for his thesis in bilirubin metabolism. He then led a research team that produces outstanding scientific contributions in aetiology and pathophysiology of upper gastrointestinal diseases. To date, he has more than 400 clinical and scientific papers, and he is a member of many distinguished learned societies. Because of his significant contributions, he has been elected as Fellow of the Academy of Medical Sciences in 1999, and Fellow of the Royal Society of Edinburgh in 2007. He is also a member of a number of national and international committees and is the chairman of a working party for the SIGN guidelines on management of *Helicobacter pylori* infection.

Among his most important discovery during his research journey is recognising the profound effects of which *Helicobacter pylori* infection can exert on gastric secretory function, and this is key to understanding the outcomes of the infection. Professor McColl is also widely known for his discovery of acid pocket. It took him more than a decade of research before the concept of acid pocket found its place in the mainstream clinical practice of gastro-oesophageal reflux disease.

More recently, he is interested with the pathophysiology and diseases of the gastro-oesophageal junction including Barrett's and cancers of the junction.

Despite his busy schedule, he is a staunch family man. He is happily married to Esther who has been supporting him all this while and likewise, his children, including Michael, David and Judith. I had the privilege to spend Christmas dinners at his home during my PhD. His loving family made me felt so much at home and I kept thinking of those happy times, each time during Christmas.

Many would be sad to hear of his pending retirement from the university including his many peers and trainees. Throughout his career, he has supervised many leading gastrointestinal physicians of today including Professor Emad El-Omar who is currently the Editor-in-Chief of Gut, a leading journal in the field of gastroenterology. During my time, I have witnessed the loss of Dr Derek Gillen, also a brilliant student and a colleague that Professor McColl was greatly fond of, and to many including myself, Dr Gillen was a highly competent clinician but also had a research mind that could possibly match Professor McColl. Equally sad to see his retirement, would include his many staff who have followed him for many years including Dorothy, Angela and Dr Mohammad Derakhshan.

Nevertheless, this year's MSGH distinguished oration befits a man who has brought significant scientific contributions to the field of gastroenterology, and we are truly honoured to have Professor McColl again, to grace our scientific congress in 2016.

13TH PANIR CHELVAM MEMORIAL LECTURER – DR JOHN MONSON

Citation by Dr Mohamed Akhtar Qureshi

Colorectal Surgery - Less Invasive, More Effective?

Professor John R T Monson is the Executive Director of Colorectal Surgery, Florida Hospital System in Orlando, Florida. He is also a Professor of Surgery at the University of Central Florida, College of Medicine. Professor Monson is a board-certified colon and rectal surgeon and fellowship trained in surgical oncology, vascular surgery, and colon and rectal surgery.

Professor Monson, who was born in Dublin, Ireland, graduated from Trinity College Dublin in 1979, and subsequently trained in Ireland and the United Kingdom. He served as a Professor of Surgery, Head of the Academic Surgical Unit, and Deputy Head of the School of Medicine at the University of Hull in England, between 1993 and 2008, before relocating to Rochester, New York, as Chief of the University of Rochester, School of Medicine Division of Colorectal Surgery and Vice-Chair of its Department of Surgery in 2008. He was also the Director of the Surgical Health Outcomes and Research Enterprise (SHORE), as well as Vice-Chairman for Research.

Professor Monson has strong links with Malaysia having trained several of the senior colorectal surgeons in Malaysia. He has on several occasions, held live surgical workshops for the benefit of local trainees. He is also an external examiner to Penang Medical College and International Medical University, here in Malaysia.

Professor Monson, whose areas of expertise include the use of minimally invasive technologies in colorectal cancer treatment including Transanal Endoscopic Microsurgery (TEMS) laparoscopy and robotic surgery. Professor Monson is credited with leading the development of laparoscopic colorectal surgery in the United Kingdom, and has been involved in basic research into a broad range of cancer-related areas and qualitative assessments of decision-making in cancer care. Professor Monson led the development of laparoscopic colorectal surgery in the United Kingdom since 1990, and was the founding Chair of the UK's National Training Programme. A lecturer and award-winning author of more than 300 peer-reviewed papers and over 100 book chapters, he is a former Vice-President of the British Association of Surgical Oncology, and served on the Executive Council of the Association of Coloproctology of Great Britain and Ireland. He was President of MIRCS (Multi-disciplinary International Rectal Cancer Society) and is the current Vice-President of the OSTRiCh Consortium on Rectal Cancer.

Professor Monson is a Fellow of the American Society of Colon and Rectal Surgeons and is currently Chair of the ASCRS Research Committee, and a member of the Foundation Board. He is also currently a member of the Rectal Cancer Coordinating Committee and Membership Committee having previously served on the Standards and Public Relations Committees. He is a Section Editor for Diseases of Colon and Rectum and serves on multiple editorial boards including Annals of Surgery and the Journal of Gastrointestinal Surgery. He is a Fellow of all Four Royal Colleges of Surgeons in the British Isles and is an Honorary Fellow of the ASGBI and the Society of University Surgeons.

His current research continues to focus on healthcare delivery and the implementation of change within healthcare systems with a particular emphasis on cancer care. In addition, he leads the implementation of the National Accreditation Program for Rectal Cancer and is a sitting member of the Commission on Cancer.

MALAYSIAN SOCIETY OF GASTROENTEROLOGY & HEPATOLOGY LIFETIME ACHIEVEMENT AWARD 2016

Citation by Dato' Dr Tan Huck Joo

Professor Dato' Dr Goh Khean Lee is a renowned figure in the world of gastroenterology both locally and internationally. His achievement in the field has been unmatched. Professor Goh is Professor at the University of Malaya and Senior Consultant at the University Malaya Medical Centre for the last 36 years. He had been the Head of Department of Medicine from 1998-2004, a member of University Senate from 2003-2006, Head and Dean of the Health and Translational Medicine Cluster (equivalent to dean of a faculty) and Head of the Division of Gastroenterology and Hepatology and GI Endoscopy Unit since 1996, at UMMC.

Professor Goh studied medicine at the University of Malaya and did his advanced training at the Royal Infirmary Glasgow in 1987, and the Academic Medical Centre at the University of Amsterdam in 1991, under Professors Guido Tytgat and Kees Huibregtse

Professor Goh has been instrumental in stimulating and catalysing research in Gastroenterology and Hepatology in this country. He has supervised and examined many doctoral theses (MD and PhD) both within and outside the University of Malaya. He is a mentor to many gastroenterologists in this country and in the region.

Under his capable leadership, Professor Goh established the GI unit as one of the best in the country with international reputation. In fact in 2008, the endoscopy unit at UMMC was awarded OMED Centre of Excellence by the World Organization of Digestive Endoscopy which has now been extended in 2015 to 2020. It is the only centre in Malaysia and one of only four centres in Asia which has been accorded this prestigious honour. The GI Endoscopy Unit is now a highly sought-after training centre, both locally and internationally.

Professor Goh was one of the founding members of MSGH in 1995, where he served as Secretary-General and then President in 1996-1997. He has served as the Chairman of Specialist Sub-committee for credentialing in gastroenterology and hepatology of the National Specialist Register, Malaysia, from 2004-2015.

Through his leadership as Organising Chairman and Scientific Chairman over the past 15 years, the MSGH has organised some top-class annual scientific meetings. This culminated with Malaysia hosting the 9th Asian Pacific Digestive Week in Kuala Lumpur in 2010, with Professor Goh as the President and the Organising Chairman. This meeting has been considered by many, to be the best APDW meeting organised so far.

In tandem with these meetings, from 1993, he has organised under the auspices of the MSGH and the University of Malaya, the annual International Therapeutic live endoscopy workshops, which is now regarded as one of the best organised endoscopy workshops in the world.

Professor Goh is currently the President of the Asia Pacific Digestive Week Federation which organises this premier annual scientific meeting in Asia Pacific (the APDW). He was the President of the Asian Pacific Association of Gastroenterology from 2010-2014. He was also Vice-President of the World Gastroenterology Organization from 2011-2015. He is the first Asian to hold such high position in WGO. In addition, he is also the Chairman of the Journal of Gastroenterology and

Hepatology Foundation. He is of course, also the recipient of the prestigious Merdeka Award in 2011, in recognition of his outstanding achievements and contribution in elevating the study and practice of gastroenterology and hepatology in Malaysia to global standards. He was also awarded the prestigious ASGE Crystal Award for International Service in 2014, for his contribution to GI Endoscopy internationally. Professor Goh is also Editor Emeritus of Journal of Gastroenterology and Hepatology.

Professor Goh has published 314 peer-reviewed papers in international journals, eight book chapters and 387 abstracts. One of the few academicians with a doctoral degree, his thesis on "*Helicobacter pylori* in Malaysia" is considered seminal work and has been widely cited throughout the world. He continues to publish consistently and was awarded the prize of "highest impact paper in a journal in 2008" by the University of Malaya. He has the highest number of publications in the biomedical field in Malaysia with papers in high impact journals such as the American Journal of Gastroenterology Gastrointestinal Endoscopy and Gut.

He is a member of several important international Consensus panels - the Maastricht 2, 3 and 4 consensus panels on *Helicobacter pylori*, Asian Pacific Consensus panels on *Helicobacter pylori* in 1997 and 2008, Gastroesophageal Reflux Disease (2002, 2005, and 2014), Colorectal Cancer Screening (2008 and 2012), NAFLD in 2008, and Upper Gastrointestinal Bleeding in 2009.

Professor Goh currently still sits in the important "Guidelines" and "Publication" sub-committee of the World Gastroenterology Organization and is a member of the review committee and co-author of the world practice guidelines on colorectal cancer screening (2006, 2010) and *Helicobacter pylori* (2006, 2009), HCV infection (2012), and liver cancer (2013). He has been invited as Visiting Professor at the University of Arizona, USA, and at the University of Magdeburg, Germany. He has also been an invited speaker for numerous international meetings including the World Congresses of Gastroenterology, APDW, and as Faculty for several live endoscopy workshops including in Hong Kong, Saudi Arabia, India, Germany, China, Japan, USA and Brazil.

Professor Goh is happily married to Su Lin, a Consultant Ophthalmologist. They have three children, two daughters, Li Yen, a Trainee Ophthalmologist in London, Li Syuen who is finishing her chemical engineering degree in London, and son, Li Han, who is currently a medical student in Australia. Professor Goh enjoys reading history and playing tennis and badminton with his children.

After 36 long years of outstanding service and achievement, Professor Goh is finally retiring from public service to embark on a new journey, a new chapter of his life.

PROGRAMME AT A GLANCE

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Daily Programme – 22ND JULY 2016, FRIDAY

0730 – 0820 Registration

0820 – 0830 Opening Remarks
by **Dr Mohamed Akhtar Qureshi**, MSGH President & Organising Chairman, GUT 2016

0830 – 0950 **SYMPOSIUM 1 – IBD** Supported by Johnson & Johnson — *SABAH ROOM*

Chairpersons: Ida Normiha Hilmi, Muhammad Radzi Abu Hassan

Crohn's disease - New treatment options for a difficult disease [pg 32]
Ng Siew Chien

Current treatment paradigm and outcome of early biologics for ulcerative colitis [pg 32]
John Marshall

Surgical challenges in Crohn's disease
John Monson

0950 – 1030 **LECTURE 1 – 16TH MSGH ORATION** — *SABAH ROOM*

Chairperson: Jayaram Menon

H. pylori and the pathophysiology of gastroduodenal and oesophageal disease [pg 33]
Kenneth McColl
Citation by Lee Yeong Yeh

1030 – 1040 Launching of the Algorithm of IBD

1040 – 1110 TEA

1110 – 1230 **BEST PAPER AWARD PRESENTATIONS**

Chairpersons: Mohamed Akhtar Qureshi, Tan Huck Joo

1230 – 1330 **Lunch Satellite Symposium** (Abbvie) — *SABAH ROOM*

Chairperson: Tan Soek Siam

SVR: What it means in real world [pg 33]
Ashley Brown

1330 – 1430 LUNCH / FRIDAY PRAYERS

1430 – 1550 **CASE DISCUSSION**

Chairpersons: Raja Affendi Raja Ali
To be presented by: Rafiz Abdul Rani

Inflammatory Bowel Disease
Panel: John Marshall, John Monson, Ng Siew Chien

1550 – 1630 **LECTURE 2** Supported by AstraZeneca — *SABAH ROOM*

Chairpersons: Ahmad Shukri Md Salleh, Ramesh Gurunathan

Long term PPI - Should we be concerned? [pg 34]
Peter Katelaris

1630 – 1730 **Tea Satellite Symposium** (Takeda) — *SABAH ROOM*

Chairperson: Goh Khean Lee

WGO 2015 Global perspective on Gastroesophageal Reflux Disease (GERD)
Justin Wu Che Yuan

1930 – 2230 **FACULTY / APPRECIATION DINNER** (By invitation only) — *SHANG PALACE, SHANGRI-LA HOTEL, KUALA LUMPUR*

Daily Programme – 23RD JULY 2016, SATURDAY

0730 – 0820 **MEET-THE-EXPERT BREAKFAST SESSIONS** (Concurrent)

1. Should I be worried about fatty liver in my patients? — *SELANGOR 1 ROOM*
   Vincent Wong Wai Sun
   Moderator: Sanjiv Mahadeva
2. Biologicals - Escalating and de-escalating therapy? — *PERAK ROOM*
   John Marshall
   Moderator: Shanthi Palaniappan
3. Personal tips in colorectal surgery — *KELANTAN ROOM*
   John Monson
   Moderator: Ismail Sagap

0820 – 0950 **CASE DISCUSSION**

Chairperson: Tan Soek Siam

Liver

Panel: Ashley Brown, London Lucien Ooi Peng-Jin, Anil Arora, James Fung Yan Yue

0950 – 1030 **LECTURE 3 – 13TH PANIR CHELVAM MEMORIAL LECTURE** — *SABAH ROOM*

Chairperson: P Kandasami

Colorectal surgery - Less invasive, more effective?
John Monson
Citation by Mohamed Akhtar Qureshi

1030 – 1100 LIFETIME ACHIEVEMENT AWARD PRESENTATION
Professor Dato' Dr Goh Khean Lee
Citation by Tan Huck Joo
Presentation of Award by **Mohamed Akhtar Qureshi**, MSGH President
Acceptance Speech by **Goh Khean Lee**

1100 – 1120 TEA

1120 – 1240 **SYMPOSIUM 2 – FGID** — *SABAH ROOM*

Chairpersons: Sanjiv Mahadeva, Tee Hoi Poh

New understanding of visceral hypersensitivity in functional gastrointestinal disorders (FGID) [pg 34]
Sutep Gonlachanvit

Reflux disease not responding to PPI [pg 35]
Kenneth McColl

Therapeutic consideration in intestinal dysbiosis [pg 35]
Justin Wu Che Yuan

1240 – 1340 **Lunch Satellite Symposium** (Abbott) — *SABAH ROOM*

Chairperson: Tan Huck Joo

Management of alcoholic hepatitis - Role of SAMe
Anil Arora

Management of drug induced liver injury - Focus on new evidence with SAMe
Oksana M Drapkina

1330 – 1420 Official Poster Round

1340 – 1440 LUNCH

1440 – 1600 **SYMPOSIUM 3 – NASH** *SABAH ROOM*

Chairpersons: Chan Wah Keong, Hamizah Razlan

High risk populations: To screen or not to screen? [pg 36]
Vincent Wong Wai Sun

Therapeutic approach for NASH patients
Anil Arora

NAFLD patients with advanced fibrosis - Treatment strategy [pg 37]
Sombat Treeprasertsuk

1600 – 1700 **Tea Satellite Symposium** (Bayer) *SABAH ROOM*

Chairperson: Goh Khean Lee

Understanding the role of evidence-based targeted therapy in HCC
Huang Yi-Hsiang

1700 – 1830 **MSGH Annual General Meeting**

Daily Programme– 24TH JULY 2016, SUNDAY

0730 – 0820 **MEET-THE-EXPERT BREAKFAST SESSIONS** (Concurrent)

4. Targeted therapy for HCC - Who do I choose and what do I look out for? — *SELANGOR 1 ROOM*
   Huang Yi-Hsiang
   Moderator: Hoe Chee Hoong
5. Who should I refer for liver transplantation? — *PERAK ROOM*
   James Fung Yan Yue
   Moderator: Koh Peng Soon

0820 – 0950 **SYMPOSIUM 4 – VIRAL HEPATITIS** — *SABAH ROOM*

Chairperson: Rosemi Bin Salleh

Hepatitis C - Dawn of a new era in treatment [pg 38]
Ashley Brown

Treatment to eradicate hepatitis B - Is there light at the end of the tunnel? [pg 38]
Lai Ching-Lung

0950 – 1030 **LECTURE 4** Supported by Ferring — *SABAH ROOM*

Chairpersons: Haniza Omar, Maylene Kok

Managing complications of liver cirrhosis and portal hypertension – Variceal bleeding and hepatorenal syndrome [pg 39]
James Fung Yan Yue

1030 – 1100 TEA

1100 – 1220 **SYMPOSIUM 5 – HCC** — *SABAH ROOM*

Chairpersons: Manisekar Subramaniam, Yoong Boon Koon

Selection of HCC patients for liver resections [pg 39]
London Lucien Ooi Peng-Jin

Therapeutic approach to liver cancer - What do we have and what can we do for our patients? [pg 40]
Huang Yi-Hsiang

Liver transplant for hepatocellular carcinoma - Is this the best option? [pg 40]
James Fung Yan Yue

1220 – 1320 **Lunch Satellite Symposium** (LF Asia) — *SABAH ROOM*

Chairperson: Goh Khean Lee

Simplifying hepatitis C treatment - Myth or reality?
Lai Ching-Lung

1320 – 1420 LUNCH

MODERATORS / CHAIRPERSONS

AHMAD SHUKRI MD SALLEH
Hospital Sultanah Nur Zahirah
Kuala Terengganu, Terengganu

CHAN WAH KEONG
University Malaya Medical Centre, Kuala Lumpur

GOH KHEAN LEE
University Malaya Medical Centre, Kuala Lumpur

HAMIZAH RAZLAN
Ampang Puteri Specialists Hospital, Selangor

HANIZA OMAR
Hospital Selayang, Batu Caves, Selangor

HOE CHEE HOONG
Penang Hospital, Penang

IDA NORMIHA HILMI
University Malaya Medical Centre, Kuala Lumpur

ISMAIL SAGAP
Universiti Kebangsaan Malaysia Medical Centre
Kuala Lumpur

JAYARAM MENON
Hospital Queen Elizabeth, Kota Kinabalu, Sabah

P KANDASAMI
International Medical University, Kuala Lumpur

KOH PENG SOON
University of Malaya, Kuala Lumpur

MAYLENE KOK
Sultanah Aminah Hospital, Johor Bahru, Johor

MANISEKAR SUBRAMANIAM
Hospital Sultanah Bahiyah, Alor Setar, Kedah

MOHAMED AKHTAR QURESHI
Sunway Medical Centre, Petaling Jaya, Selangor

MUHAMMAD RADZI ABU HASSAN
Hospital Sultanah Bahiyah, Alor Setar, Kedah

RAJA AFFENDI RAJA ALI
Universiti Kebangsaan Malaysia Medical Centre
Kuala Lumpur

RAMESH GURUNATHAN
Sunway Medical Centre, Petaling Jaya, Selangor

ROSEMI BIN SALLEH
Hospital Raja Perempuan Zainab II, Kota Bharu
Kelantan

SANJIV MAHADEVA
University Malaya Medical Centre, Kuala Lumpur

SHANTHI PALANIAPPAN
Pantai Hospital Kuala Lumpur, Kuala Lumpur

TAN HUCK JOO
Sunway Medical Centre, Petaling Jaya, Selangor

TAN SOEK SIAM
Hospital Selayang, Batu Caves, Selangor

TEE HOI POH
Hospital Tengku Ampuan Afzan, Kuantan, Pahang

YOONG BOON KOON
University Malaya Medical Centre, Kuala Lumpur

FACULTY BIO-DATA

ANIL ARORA

Dr Anil Arora is a Gastroenterologist and Chairman of Department of Gastroenterology and Hepatology of Sir Ganga Ram Hospital, Lahore, India. With over 90 publications, his major contributions have been in relation to the role of achlorhydria in stopping and preventing peptic ulcer bleeding, and diagnosis and management of Budd-Chiari syndrome. Besides being skilled in endoscopy, he is known for his works in viral hepatitis, and is currently a senior member of the transplant team in the hospital.

ASHLEY BROWN

Dr Brown is Consultant Hepatologist at St Mary's and Hammersmith Hospitals in London, United Kingdom, and Adjunct Reader in Medicine at Imperial College London. Dr Brown completed his medical studies at the University of Liverpool, UK, and undertook post-graduate training in liver disease at the University of Newcastle-upon-Tyne. Dr Brown has a major research interest in viral hepatitis, and he has served as chief investigator or principal investigator on a large number of clinical trials of novel hepatitis therapies. He is a Fellow of the Royal College of Physicians, a committee member of the British Association for the Study of the Liver, and is a former Chairman of the British Viral Hepatitis Group.

OKSANA M DRAPKINA

Dr Drapkina is Deputy Director for research and clinical work of FGBU "SRCPM" Russian Ministry of Health, Professor of Propaedeutic Department of the First Moscow State Medical University named after I.M. Sechenov, as well as Professor of the Internal Medicine Propaedeutic Clinic named after Academician VH Vasilenko of Moscow Medical Academy named after Sechenov (Gastroenterology and Hepatology Department), and Secretary of the Inter-departmental Scientific Council.

JAMES FUNG YAN YUE

Dr James Fung is a Consultant at the Department of Medicine, and a Transplant Hepatologist at the Liver Transplant Centre, Queen Mary Hospital, Hong Kong. He was awarded the Fellow of the Royal Australasian College of Physicians in 2005, and Fellow of the Hong Kong College of Physicians in 2008. In 2011, he attained his Doctor of Medicine. He has published widely with research interests in viral hepatitis, liver fibrosis, advanced liver disease, hepatocellular carcinoma, and liver transplantation.

FACULTY BIO-DATA (CONT'D)

SUTEP GONLACHANVIT

Dr Gonlachanvit is Professor, Chief of Gastrointestinal Motility Research Unit, and Vice-President and Chair of Medical and Research Affair, King Chulalongkorn Memorial Hospital in Bangkok, Thailand. He is the current President of the Asian Neurogastroenterology and Motility Association, a founder member of the Thai Neurogastroenterology and Motility Society, a member of the Rome Asian working team and associate editor of Journal of Neurogastroenterology and Motility.

HUANG YI-HSIANG

Dr Huang is Professor at the Institute of Clinical Medicine, National Ying-Ming University in Taipei, Taiwan. He is also an attending physician in the Division of Gastroenterology and Hepatology at the Taipei Veterans General Hospital. Dr Huang was a post-doctoral fellow at the Vaccine Branch, National Institutes of Health in Bethesda, United States. Dr Huang is an academic editor of *PLoS One* and an editorial board member of *Liver International*.

PETER KATELARIS

Dr Katelaris is an Associate Professor and Senior Consultant Gastroenterologist at Concord Hospital, University of Sydney. His research interests include gastroesophageal reflux disease and *Helicobacter pylori* infection. He has co-authored the Australian, World Gastroenterology Organisation and Asia Pacific Gastroesophageal Reflux Disease Guidelines, and was a member of the Kyoto International Summit on Gastritis and *Helicobacter pylori*. He is recognised as an experienced clinician, endoscopist, researcher and educator. He has taught at all levels of medical education for many years.

LAI CHING-LUNG

Dr Lai is the Simon K Y Lee Professor in Gastroenterology and the Chair Professor of Medicine and Hepatology at the Department of Medicine, University of Hong Kong, his alma-mater. He works extensively on various aspects of the hepatitis B virus, including molecular virology, natural history, treatment and its prevention, and more recently, chronic hepatitis C. He has published over 470 widely cited publications and is one of the top scientists in the field of chronic hepatitis B infection. He was invited to give the Leon Schiff State-of-the-Art Lecture at the 2005 annual meeting of the American Association for the Study of Liver Diseases.

FACULTY BIO-DATA (CONT'D)

JOHN MARSHALL

Dr Marshall is Professor of Medicine at McMaster University and Chief of Service for Gastroenterology at Hamilton Health Sciences in Hamilton, Ontario, Canada. He completed his MD at Queen's University, and MSc in Clinical Epidemiology at McMaster. He is Full Member of the Farncombe Family Digestive Health Research Institute, Editor-in-Chief of *Canadian Journal of Gastroenterology and Hepatology,* Associate Editor of *ACP Journal Club* and *Evidence-Based Medicine,* and Editorial Board Member for *Journal of Crohn's and Colitis* and *Clinical Gastroenterology and Hepatology.*

KENNETH McCOLL

Dr McColl obtained his medical degree from the University of Glasgow in 1974, and then undertook training in both General Internal Medicine and Gastroenterology. In 1984, he was appointed Consultant Physician in Gastroenterology in the Western Infirmary, Glasgow, and in 1992, he was appointed Professor of Gastroenterology in the University of Glasgow. Dr McColl has led an active research group focusing on the aetiology and pathophysiology of upper gastrointestinal disease. He is a Fellow of the Academy of Medical Sciences and a Fellow of the Royal Society of Edinburgh, Scotland.

JOHN MONSON

Dr Monson is the Executive Director of Colorectal Surgery, Florida Hospital System in Orlando, Florida. He is also a Professor of Surgery at the University of Central Florida, College of Medicine. He is an expert of minimally invasive technologies in colorectal cancer treatment. He authored more than 300 peer-reviewed papers and over 100 book chapters. He was former Vice-President of the British Association of Surgical Oncology, and served on the Executive Council of the Association of Coloproctology of Great Britain and Ireland.

NG SIEW CHIEN

Dr Ng is Associate Professor at the Department of Medicine and Therapeutics, Chinese University of Hong Kong. Her research interest includes inflammatory bowel disease, gut microbiota and colorectal cancer. She has published over 120 original articles in high impact international journals. She is President of the Hong Kong IBD Society, Management Committee of the International IBD Genetics Consortium and Deputy Secretary of the International Organization of Inflammatory Bowel Disease. Dr Ng has been recognised worldwide with several national and international awards.

FACULTY BIO-DATA (CONT'D)

LONDON LUCIEN OOI PENG-JIN

Dr London Lucien Ooi is Professor and Senior Hepatobiliary Surgical Oncologist. He is a keen researcher with more than 200 publications and 14 books/chapters to his credit. He has a doctorate (MD) for research on hepatocellular carcinoma, and his interests include biomarkers, hepatocellular carcinoma therapeutics, and medical technology devices. As an active teacher, he has trained surgeons in advanced hepatobiliary surgery from around the world, and is also an international examiner in some countries. He sits on numerous national and international committees, advisory panels, research, editorial and funding agency boards.

SOMBAT TREEPRASERTSUK

Dr Treeprasertsuk is Professor at the Chulalongkorn University, Bangkok, Thailand. He obtained his medical degree from the Mahidol University, Thailand, in 1997, and he was Assistant Professor at faculty of Tropical Medicine, Mahidol University, in 2000, before moving to Chulalongkorn University, in 2005. In 2008-2010, he was a research fellow at the Mayo Clinic, Rochester, United States. In 2012, he received his PhD degree from Chulalongkorn University. In 2015, he was appointed as Professor and Deputy Chair of Department of Medicine. Dr Sombat has led an active research group focusing on the non-alcoholic fatty liver diseases and cirrhotic complications. Currently, at least 100 of his publications, abstracts and book chapters have been cited.

VINCENT WONG WAI SUN

Dr Vincent Wong is Professor at the Department of Medicine and Therapeutics of The Chinese University of Hong Kong. He is currently, the President of the Hong Kong Association for the Study of Liver Diseases and an associate editor of *Liver International*. His research focuses on viral hepatitis and non-alcoholic fatty liver disease. He has authored over 230 articles in international medical journals. His papers have been cited by various guidelines, and his latest h-index is 50.

JUSTIN WU CHE YUAN

Dr Justin Wu is Professor and Associate Dean (Development) of Faculty of Medicine, The Chinese University of Hong Kong. He specialises in functional gastrointestinal diseases, gastroesophageal reflux disease and gastrointestinal motility with over 200 publications. He is the Scientific Chairman of Asian Neurogastroenterology and Motility Association, Treasurer of Asia-Pacific Association of Gastroenterology, and the President of Asia-Pacific Digestive Week 2017, in Hong Kong. He is also a Managing Editor of Journal of Gastroenterology and Hepatology and has served as an associate editor for the American Journal of Gastroenterology.

MSGH ANNUAL SCIENTIFIC MEETINGS

&

ENDOSCOPY WORKSHOPS

THE PROUD TRADITION OF THE

MALAYSIAN SOCIETY OF GASTROENTEROLOGY AND HEPATOLOGY

ANNUAL THERAPEUTIC ENDOSCOPY WORKSHOPS – "ENDOSCOPY"

(Organised by the Malaysian Society of Gastroenterology and Hepatology in collaboration with the University of Malaya)

EVENT	FACULTY	DATE
Difficult ERCP- "The Master's Approach"	Kees Huibregtse (Amsterdam, The Netherlands)	19th August 1993
Endoscopic Ultrasonography	TL Tio (Washington, USA)	26th July 1994
ERCP- "Basic Skills, Finer Points and New Techniques"	Kees Huibregtse (Amsterdam, The Netherlands)	25th August 1994
Practical Points in Therapeutic Endoscopy	Nib Soehendra (Hamburg, Germany)	6th December 1994
Therapeutic Endoscopy Workshop (In conjunction with Island Hospital, Penang, Malaysia)	Nib Soehendra (Hamburg, Germany) Kees Huibregtse (Amsterdam, Netherlands)	22nd July 1997
Lasers in Gastroenterology	R Leicester (London, United Kingdom)	13th August 1997
GI Endoscopy Nurses Workshop – "Setting the Standards for Practice"	Staff Members - Endoscopy Unit, University Hospital, Kuala Lumpur, Malaysia	30th April - 2nd May 1999
Endoscopy 2000	Sydney C S Chung (Hong Kong, China), Kenji Yasuda (Kyoto, Japan), Wang Yong-Guang (Beijing, China), Nageshwar Reddy (Hyderabad, India) *GIA Faculty:* Dorothy Wong (Hong Kong, China)	13th - 15th April 2000
Endoscopy 2001 – "A Master Class in Therapeutic Endoscopy"	Nib Soehendra (Hamburg, Germany) *GIA Faculty:* Adriana Cargin (Melbourne, Australia)	14th - 15th April 2001
Endoscopy 2002 – "Enhancing Basic Skills and Developing Expertise"	Christopher Williams (London, United Kingdom), Naotaka Fujita (Sendai, Japan), Joseph Leung (Sacramento, USA), Kees Huibregtse (Amsterdam, Netherlands) *GIA Faculty:* Diana Jones (Sydney, Australia)	5th - 7th April 2002
Endoscopy 2003 – "The Cutting Edge of GI Endoscopy"	Douglas Howell (Portland, USA), Haruhiro Inoue (Tokyo, Japan) Simon K Lo (Los Angeles, USA), Nageshwar Reddy (Hyderabad, India)	28th February - 2nd March 2003
Endoscopy 2004 – "Appreciating the Art of GI Endoscopy"	Firas Al Kawas (Washington, USA), Yoshihiro Sakai (Tokyo, Japan), Stefan Seewald (Hamburg, Germany), Joseph Sung (Hong Kong, China)	5th - 7th March 2005
Endoscopy 2005 – "Defining the Scope of Excellence"	Guido Costamagna (Rome, Italy), Shim Chan-Sup (Seoul, South Korea), K Yasuda (Kyoto, Japan), B Rembacken (Leeds, United Kingdom)	1st - 3rd April 2005
Endoscopy 2006 – "Frontiers of Therapeutic Endoscopy"	A T R Axon (Leeds, United Kingdom), James Lau (Hong Kong, China), Seo Dong-Wan (Seoul, Korea), Irving Waxman (Chicago, USA), Naohisa Yahagi (Tokyo, Japan)	14th - 16th April 2006
Endoscopy 2007 – "The Best Endoscopic Practices	Nageshwar Reddy (Hyderabad, India), Reza Shaker (Milwaukee, USA), Yusuke Saitoh (Sapporo, Japan), Stefan Seewald (Hamburg, Germany), Song Si-Young (Seoul, Korea), Mary Bong (Sydney, Australia)	13th - 15th April 2007
Endoscopy 2008 – "Seeing Better, Doing Better"	Peter B Cotton (Charleston, USA), G Ginsberg (Philadelphia, USA), H Isayama (Tokyo, Japan), S Ryozawa, (Yamaguchi, Japan), J S Byeon (Seoul, Korea), Syed Shah, (West Yorkshire, United Kingdom)	29th February, 1st - 2nd March 2008

ANNUAL THERAPEUTIC ENDOSCOPY WORKSHOPS – "ENDOSCOPY" (CONT'D)

(Organised by the Malaysian Society of Gastroenterology and Hepatology in collaboration with the University of Malaya)

EVENT	FACULTY	DATE
Endoscopy 2009 – "Exploring the Limits of Endoscopy"	Jerome D Waye (New York, USA), Kulwinder Dua (Milwaukee, USA), Amit Maydeo (Mumbai, India), H Kawamoto (Okayama,Japan), I Yasuda (Gifu, Japan), Lee Yong-Chan (Seoul, Korea), Y Sano (Kobe, Japan)	20th - 22nd March 2009
Endoscopy 2010 (organised with the APDW 2010) (In conjunction with Selayang Hospital, Kuala Lumpur, Malaysia)	Michael Bourke (Sydney, Australia), David Carr-Locke (New York, USA), Mitsuhiro Fujishiro (Tokyo, Japan), Marc Giovannini (Marseilles-France), Takuji Gotoda (Tokyo, Japan), James Lau (Hong Kong, China), Amit Maydeo (Mumbai, India), Ibrahim Mostafa (Cairo, Egypt), Horst Neuhaus (Düsseldorf, Germany), Nageshwar Reddy (Hyderabad, India), Rungsun Reknimitr (Bangkok, Thailand), Seo Dong-Wan (Seoul, Korea), Naohisa Yahagi (Tokyo, Japan), Hironori Yamamoto (Tokyo, Japan), Kenjiro Yasuda (Kyoto, Japan)	20th - 21st September 2010
Endoscopy 2011 – "What's New and What's Good for Our Patients"	Hisao Tajiri (Tokyo, Japan), Chiu Han-Mo (Taipei, Taiwan), Arthur Kaffes (Sydney, Australia), Ho Khek-Yu (Singapore), Hiroo Imazu (Tokyo, Japan), Takao Itoi (Tokyo, Japan), Lee Dong-Ki (Seoul, Korea), Takahisa Matsuda (Tokyo, Japan), Moon Jong-Ho (Seoul, Korea)	14th - 17th April 2011
Endoscopy 2012 – "Therapeutic Endoscopy in the Global World"	Robert Hawes (Miami, USA), Hiroshi Kashida (Kinki, Japan), Lee Sang-Hyup (Seoul, Korea), Claudio Navarette (Santiago, Chile), Paulo Sakai (Sao Paulo, Brazil), Rajvinder Singh (Adelaide,Australia), Wang Hsiu-Po (Taipei, Taiwan), Kenshi Yao (Fukuoka, Japan)	30th - 31st March, 1st April 2012
Endoscopy 2013 – "Advancing the Practice of Endoscopy"	Phillip Chiu (Hong Kong, China), Lawrence Khek-Yu Ho (Singapore), Horst Neuhaus (Dusseldorf, Germany), Krish Ragunath (Nottingham, United Kingdom), Dong-Wan Seo (Seoul, Korea), Yun-Sheng Yang (Beijing, China), Ian Yusoff (Perth, Australia) *Special GIA Faculty:* Wang Ping (Shanghai, China)	12th - 14th April 2013
Endoscopy 2014 – "The Best Tips in Therapeutic Endoscopy"	Mitsuhiro Kida (Kanagawa, Japan), Gregory Ginsberg (Philadelphia, USA), Yutaka Saito (Tokyo, Japan), Jin Hong Kim (Suwon, Korea), James Y W Lau (Shatin, Hong Kong) *Special GIA Faculty:* Mary Bong (Sydney, Australia)	28th - 30th March 2014
Endoscopy 2015 – "Maintaining Quality in Endoscopy"	Christopher Khor (Singapore), Sundeep Lakhtakia (Hyderabad, India), Hiroyuki Maguchi (Sapporo, Japan), Amit Maydeo (Mumbai, India), Jong-Ho Moon (Bucheon, Korea), Roy Soetikno (Singapore and California, USA), Kenneth Wang (Rochester, Usa)	17th - 19th April 2015
Endoscopy 2016 – "Expanding the Horizons of Therapeutic Endoscopy"	Hyun-Jong Choi (Bucheon, Korea), Jacques Deviere (Brussels, Belgium), Manoel Galvao Netto (Sao Paulo, Brazil), Nageshwar Reddy (Hyderabad, India), Rungsun Reknitmitr (Bangkok, Thailand), Brian Saunders (London, UK), Shyam Varadarajulu (Orlando, USA)	8th - 10th April 2016

DISTINGUISHED ENDOSCOPY LECTURERS

NO	YEAR	ORATOR	TOPIC
1st	1999	Kees Huibregtse (Amsterdam, The Netherlands)	The Development and Use of Biliary Endoprosthesis in ERCPs
2nd	2001	Nib Soehendra (Hamburg, Germany)	A Master's Approach to Therapeutic Endoscopy
3rd	2002	Christopher Williams (London, United Kingdom)	Practical Tips and Pitfalls in Colonoscopy
4th	2003	Guido N J Tytgat (Amsterdam, The Netherlands)	The Unlimited Horizons of Therapeutic Endoscopy
5th	2004	Yoshio Sakai (Tokyo, Japan)	Development and Application of Colonoscopy
6th	2005	Guido Costamagna (Rome, Italy)	Endoscopic Management of Pancreatobiliary Diseases – State-of-the-art in 2005
7th	2006	Anthony T R Axon (Leeds, United Kingdom)	The Impact of New Technology in GI Endoscopy
8th	2007	D Nageshwar Reddy (Hyderabad, India)	Chronic Pancreatitis – Genes to Bedside
9th	2008	Peter Cotton (Charleston, USA)	Therapeutic Endoscopy – Then, Now and Maybe
10th	2009	Jerome Waye (New York, USA)	Exploring the Limits of Endoscopy
11th	2010	David L Carr-Locke (New York, USA)	Enhancing the Eye – The Future of Endoscopy
12th	2011	Hisao Tajiri (Tokyo, Japan)	Enhanced Imaging of the Gastrointestinal Tract
13th	2012	Robert Hawes (Orlando, USA)	The Current and Future Role of Endoscopic Ultrasonography in GI Practice
14th	2013	Horst Neuhaus Dusseldorf, Germany)	Viewing the Bile Duct – Recent Developments of Cholangioscopy
15th	2014	Gregory Ginsberg (Philadelphia, USA)	Future Prospects for Gastrointestinal Endoscopy
16th	2015	Kenneth Wang (Rochester, USA)	Diagnosis and Endoscopic Treatment of Barrett's Esophagus
17th	2016	Jacques Deviere (Brussels, Belgium)	Metabolic Endoscopy: Future Horizons in Therapeutic Endoscopy

ANNUAL SCIENTIFIC MEETINGS – OVERSEAS INVITED FACULTY

THE STOMACH '96 (Co-organised with the College of Surgeons)

3rd – 6th July 1996, Kuala Lumpur

Stephen G Bown	United Kingdom	Kang Jin-Yong	United Kingdom	Henry M Sue-Ling	United Kingdom
Sydney C S Chung	Hong Kong	Lam Shiu-Kum	Hong Kong	Nicholas J Talley	Australia
Teruyuki Hirota	Japan	Adrian Lee	Australia	Guido N J Tytgat	Netherlands
Richard H Hunt	Canada	Roy E Pounder	United Kingdom	Cornelis J H Van De Velde	Netherlands
David Johnston	United Kingdom	Robert H Riddell	Canada		

PENANG INTERNATIONAL TEACHING COURSE IN GASTROENTEROLOGY

(Co-organised with Penang Medical Practitioners' Society with the participation of the British Society of Gastroenterology)

23rd – 26th July 1997, Penang

Anthony Axon	United Kingdom	Dermot Kelleher	Ireland	J J Misiewicz	United Kingdom
John Dent	Australia	Fumio Konishi	Japan	James Neuberger	United Kingdom
R Hermon Dowling	United Kingdom	John Lambert	Australia	Thierry Poynard	France
Greg Holdstock	United Kingdom	Michael Larvin	United Kingdom	Jonathan Rhodes	United Kingdom
Kees Huibregtse	Netherlands	Christopher Liddle	Australia	Nib Soehendra	Germany
P W N Keeling	Ireland	Lim Seng-Gee	Singapore		

SECOND WESTERN PACIFIC HELICOBACTER CONGRESS

25th – 27th July 1998, Kota Kinabalu, Sabah

Masahiro Asaka	Japan	Richard Hunt	Canada	Pentti Sipponen	Finland
Douglas E Berg	USA	Lam Shiu-Kum	Hong Kong, China	Joseph J Y Sung	Hong Kong, China
Fock Kwong-Ming	Singapore	Adrian Lee	Australia	Rakesh Tandon	India
David Forman	United Kingdom	Peter Malfertheiner	Germany	Guido N J Tytgat	Netherlands
David Y Graham	USA	Kenneth E L McColl	Scotland	Xiao Shu-Dong	China
Stuart L Hazell	Australia	Hazel M Mitchell	Australia		

GASTROENTEROLOGY 1999

23rd – 25th July 1999, Kuala Terengganu, Terengganu

Francis K L Chan	Hong Kong, China	Mohammed Al Karawi	Saudi Arabia	Quak Seng-Hock	Singapore
Sydney S C Chung	Hong Kong, China	Mohammad Sultan Khuroo	Saudi Arabia	Nicholas J Talley	Australia
John Dent	Australia	Peter Malfertheiner	Germany	Neville D Yeomans	Australia
Rikiya Fujita	Japan	Colm O'Morain	Ireland		

GUT 2000

24th – 26th August 2000, Melaka

Anthony Axon	United Kingdom	Lim Seng-Gee	Singapore	Francis Seow-Choen	Singapore
Geoffrey C Farrell	Australia	Anthony I Morris	United Kingdom	Jose D Sollano	Philippines
Vay Liang W Go	USA	David Mutimer	United Kingdom	Guido N J Tytgat	Netherlands
Humphrey J F Hodgson	United Kingdom	Ng Han-Seong	Singapore	Michael Wolfe	USA
Peter Katelaris	Australia	Thierry Poynard	France		

GASTRO 2001 (With the participation of the American Gastroenterological Association)

5th – 8th April 2001, Kota Kinabalu, Sabah

Aziz Rani	Indonesia	Y K Joshi	India	Mahesh P Sharma	India
Chung Owyang	USA	Joseph Kolars	USA	Gurkirpal Singh	USA
Sydney S C Chung	Hong Kong, China	Koo Wen-Hsin	Singapore	Jose D Sollano	Philippines
Andrew Clouston	Australia	Edward Krawitt	USA	J L Sweeney	Australia
John Dent	Australia	Pinit Kullavanijaya	Thailand	Rakesh Tandon	India
Fock Kwong-Ming	Singapore	Lam Shiu-Kum	Hong Kong, China	Benjamin C Y Wong	Hong Kong, China
Robert N Gibson	Australia	Peter Malfertheiner	Germany	Xiao Shu-Dong	PR China
Richard Hunt	Canada	James M Scheiman	USA		

GUT 2002

27th – 30th June 2002, Penang

Chow Wan-Cheng	Singapore	Peter Katelaris	Australia	Ng Han-Seong	Singapore
Anuchit Chutaputti	Thailand	James Y W Lau	Hong Kong, China	C S Pitchumoni	USA
David Forman	United Kingdom	Tore Lind	Sweden	Herbert J Tilg	Austria
Lawrence Ho Khek-Yu	Singapore	Barry James Marshall	Australia	John Wong	Hong Kong, China

GUT 2003

28th – 31st August 2003, Kuching, Sarawak

Francis K L Chan	Hong Kong, China	Humphrey J O'Connor	Ireland	Eamonn M M Quigley	Ireland
Chang Mei-Hwei	Taiwan	Colm O'Morain	Ireland	Jose D Sollano Jr	Philippines
W G E Cooksley	Australia	Teerha Piratvisuth	Thailand	Joseph Sung	Hong Kong, China
Gwee Kok-Ann	Singapore	Roy Pounder	United Kingdom	Yeoh Khay-Guan	Singapore

GUT 2004

24th – 27th June 2004, Penang

Sydney C S Chung	Hong Kong, China	Huang Jia-Qing	China	Mario Rizzetto	Italy
Geoffrey C Farrell	Australia	Lam Shiu-Kum	Hong Kong, China	Russell W Strong	Australia
Ronnie Fass	USA	Peter W R Lee	United Kingdom	Benjamin C Y Wong	Hong Kong, China
David Fleischer	USA	Masao Omata	Japan		
Fock Kwong-Ming	Singapore	Teerha Piratvisuth	Thailand		

GUT 2005

23rd – 25th June 2005, Pulau Langkawi, Kedah

Raymond Chan Tsz-Tong	Hong Kong, China	Gerald Johannes Holtmann	Australia	Graeme Young	Australia
Meinhard Classen	Germany	Peter Malfertheiner	Germany	Yuen Man-Fung	Hong Kong, China
Anthony Goh	Singapore	Kenneth McColl	Ireland		

GUT 2006

20th – 23rd June 2006, Kuala Lumpur

Peter Gibson	Australia	Anthony Morris	United Kingdom	Francis Seow-Choen	Singapore
Lawrence Ho Khek-Yu	Singapore	Nageshwar Reddy	India	Nimish Vakil	USA
Gerald Johannes Holtmann	Germany	Ng Han-Seong	Singapore	John Wong	Hong Kong, China
Lim Seng-Gee	Singapore	Ooi Choon-Jin	Singapore		
Irvin Modlin	USA	Fred Poordad	USA		

GUT 2007

29th August – 1st September 2007, Kota Kinabalu, Sabah

Ronnie Fass	USA	Norman Marcon	USA	Nib Soehendra	Germany
Marc Giovannini	France	Amit Maydeo	India	Daniel Wong	Singapore
Robert Hawes	USA	Charlie Millson	England	Hironori Yamamoto	Japan
Richard Hunt	Canada	G V Rao	India	Yeoh Khay-Guan	Singapore
Finlay Macrae	Australia	Marcelo Silva	Argentina		

GUT 2008

21st – 24th August 2008, Kuala Lumpur

Anuchit Chutaputti	Thailand	Lawrence Ho Khek-Yu	Singapore	Govind K Makharia	India
Peter Bytzer	Sweden	Pali Hungin	United Kingdom	Prateek Sharma	USA
Henry Chan Lik-Yuen	Hong Kong, China	Rupert Leong	Australia	Rajvinder Singh	Australia
Sydney C S Chung	Hong Kong, China	Davide Lomanto	Singapore	Mitchell Shiffman	USA
David Y Graham	USA	Lui Hock-Foong	Singapore	Sundeep Punamiya	Singapore

GUT 2009

14th to 16th August 2009, Pulau Langkawi, Kedah

Geoffrey Farrell	Australia	Lim Seng-Gee	Singapore	Joseph Sung Jao-Yiu	Hong Kong, China
Fock Kwong-Ming	Singapore	Lo Chung-Mau	Hong Kong, China	Daniel Wong Wai-Yan	United Kingdom
Peter R Galle	Germany	Irvin Modlin	USA	Yeoh Khay-Guan	Singapore
Christopher Khor	Singapore	Fabio Pace	Italy		
George K K Lau	Hong Kong, China	Rungsun Rerknimitr	Thailand		

APDW 2010 (Incorporating GUT 2010 & Endoscopy 2010)

19th to 22nd September 2010, Kuala Lumpur Convention Centre, Kuala Lumpur

Subrat Kumar Acharya	India	Hiroyuki Isayama	Japan	Eamonn Quigley	Ireland
Deepak Amarapurkar	India	Takao Itoi	Japan	Shanmugarajah Rajendra	Australia
Ang Tiing-Leong	Singapore	Derek Jewell	United Kingdom	Gurudu Venkat Rao	India
John Atherton	United Kingdom	Jia Ji-Dong	China	Nageshwar Reddy	India
Anthony Axon	United Kingdom	Utom Kachintorn	Thailand	Rungsun Rerknimitr	Thailand
Deepak Bhasin	India	Hiroshi Kashida	Japan	Jean Francois Rey	France
Henry J Binder	USA	Peter Katelaris	Australia	Shomei Ryozawa	Japan
Mary Bong	Australia	Takashi Kawai	Japan	Yutaka Saito	Japan
Michael Bourke	Australia	Christopher Khor Jen-Lock	Singapore	Shiv Sarin	India
Marco Bruno	The Netherlands	Nayoung Kim	Korea	Wolff Schmiegel	Germany
David Carr-Locke	USA	Seigo Kitano	Japan	Juergen Schoelmerich	Germany
Ashok Chacko	India	Sriram Krishnan	USA	See Teik-Choon	United Kingdom
Henry Chan Lik-Yuen	Hong Kong, China	Shin-ei Kudo	Japan	Seo Dong-Wan	Korea
Francis Chan Ka-Leung	Hong Kong, China	Ashish Kumar	India	Francis Seow-Choen	Singapore
Adarsh Chaudhary	India	George Lau	Hong Kong, China	Prateek Sharma	USA
Yogesh Chawla	India	James Lau Yun-Wong	Hong Kong, China	Shim Chan-Sup	Korea
Yang Chen	USA	Rupert Leong	Australia	Hiroshi Shimada	Japan
Chen Min-Hu	China	Leung Wai-Keung	Hong Kong, China	Jose Sollano	Philippines
Philip Chiu	Hong Kong, China	Lim Seng-Gee	Singapore	Eduard Stange	Germany
Pierce Chow	Singapore	Lin Jaw-Town	Taiwan	Russell W Strong	Australia
Chow Wan-Cheng	Singapore	Liu Chen-Hua	Taiwan	Kentaro Sugano	Japan
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Richard Hunt	Canada	Qian Jia-Ming	China	Qi Zhu	China

GUT 2011

27th to 29th May 2011, Kuala Lumpur

Ling Khoon-Lin	Singapore	Chan See-Ching	Hong Kong, China	See Teik-Choon	United Kingdom
Luigi Bolondi	Italy	Colm O'Morain	Ireland	Kao Jia-Horng	Taiwan
Lui Hock-Foong	Singapore	Philip Chiu Wai-Yan	Hong Kong, China	Yeoh Khay-Guan	Singapore
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GUT 2012

29th June to 1st July 2012, Melaka

Henry Chan Lik-Yuen	Hong Kong, China	James Y W Lau	Hong Kong, China	Morris Sherman	Canada
Emad El-Omar	USA	Francesco Marotta	Italy	Shaw Somers	United Kingdom
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GUT 2013

23rd to 25th August 2013, Penang

Alan Barkun	Canada	David Kwon	Korea	Takeshi Sano	Japan
Francis Chan	Hong Kong, China	Kenneth EL McColl	United Kingdom	Francis Seow-Choen	Singapore
Chien Rong-Nan	Taiwan	Ng Siew-Chien	Hong Kong, China	Vijay Shah	USA
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GUT 2014 & ECCO EDUCATIONAL WORKSHOP

22nd to 24th August 2014, Kuala Lumpur

Adarsh Chaudhary	India	Nancy Leung	United Kingdom	Stephan Vavricka	Switzerland
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GUT 2015

21st to 23rd August 2015, Johor Bahru, Johor

Francis Chan Ka-Leung	Hong Kong, China	Leung Wai Keung	Hong Kong, China	Rajesh Sainani	India
Yogesh Chawla	India	Lim Jit Fong	Singapore	Teik-Choon See	United Kingdom
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MSGH ORATION LECTURERS

NO	YEAR	ORATOR	TOPIC
1st	2001	P Kandasami Kuala Lumpur, Malaysia	Gastroenterology in Malaysia
2nd	2002	Barry J Marshall Perth, Australia	*Helicobacter pylori*: How it all came about and where do we go from here?
3rd	2003	Guido J Tytgat Amsterdam, The Netherlands	Future Developments in Gastroenterology
4th	2004	Lam Shiu-Kum Hong Kong, China	Pathogenesis of Gastric Cancer – A Unifying Concept
5th	2005	Meinhard Classen Munich, Germany	GI Cancer – The Global Burden in the New Millennium
6th	2006	John Wong Hong Kong, China	Multi-Disciplinary Treatment in Esophageal Cancer: The Price of Failure
7th	2007	Norman Marcon Toronto, Canada	New Optical Technologies for Early Detection of Dysplasia
8th	2008	Sydney Chung Hong Kong, China	Ulcer Bleeding: What you really want to know
9th	2009	Geoffrey Farrell Canberra, Australia	Battling the Bulge in Asia – Implications for Gastroenterologists
10th	2010	Nicholas Joseph Talley Newcastle, Australia	New Insights into the Aetiopathogenesis of Functional Dyspepsia
11th	2011	Colm O'Morain Dublin, Ireland	Colorectal Cancer – The Emerging Cancer in the 21st Century
12th	2012	Richard Kozarek Seattle, USA	Minimally Invasive/Interventional Gastroenterology: Where Have We Been? Where Are We Going?
13th	2013	Goh Khean Lee Kuala Lumpur, Malaysia	Asia at the Crossroads: Changing Patterns and Emerging Diseases
14th	2014	Patrick Kamath Minnesota, USA	Insights into Optimal Management of End Stage Liver Disease - A Continuing Challenge
15th	2015	Kentaro Sugano Tokyo, Japan	*Helicobacter pylori* and Gastric Cancer - A Balanced View

PANIR CHELVAM MEMORIAL LECTURERS

NO	YEAR	ORATOR	TOPIC
1st	2004	Mohd Ismail Merican Kuala Lumpur, Malaysia	Treatment of Chronic Viral Hepatitis in the Asia-Pacific Region: Realities and Practical Solutions
2nd	2005	Peter Malfertheiner Magdeburg, Germany	Diagnosis and Management of Pancreatic Cancer
3rd	2006	Nageshwar Reddy Hyderabad, India	GI Endoscopy in India – Developement and Lessons for the Future
4th	2007	Richard Hunt Hamilton, Canada	Evidence-based Medicine in the Real World
5th	2008	Pali Hungin Durham, United Kingdom	Plausible Solutions for Impossible Problems
6th	2009	Fock Kwong-Ming Singapore	Lower GI Bleeding – Epidemiology and Management
7th	2010	Joseph J Y Sung Hong Kong, China	The Future Role of the Gastroenterologist in Digestive Oncology
8th	2011	Kang Jin-Yong London, United Kingdom	East-West Differences in Upper GI Diseases
9th	2012	Emad El-Omar Aberdeen, United Kingdom	Role of Chronic Inflammation in GI Cancer
10th	2013	Michael Kamm Melbourne, Australia	Achieving the Balance between Drug Therapy and Surgery in Inflammatory Bowel Disease
11th	2014	John A Windsor Auckland, New Zealand	Progress with Acute Pancreatitis – Millstones and Milestones
12th	2015	Yogesh Chawla Jabalpur, India	Non Cirrhotic Portal Hypertension

CONFERENCE INFORMATION

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GUT 2016
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The registration hours are:

21st August 2016 (Thursday)	1600 to 1830 hrs
22nd August 2016 (Friday)	0730 to 1700 hrs
23rd August 2016 (Saturday)	0730 to 1700 hrs
24th August 2016 (Sunday)	0730 to 1100 hrs

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21st August 2016 (Thursday)	1600 to 1830 hrs
22nd August 2016 (Friday)	0730 to 1700 hrs
23rd August 2016 (Saturday)	0730 to 1700 hrs
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All presentations will be deleted from the conference computers after the presentations are over.

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Posters will be displayed at the foyer from 0800 hrs on 22nd August 2016 till 1200 hrs on 24th August 2016.

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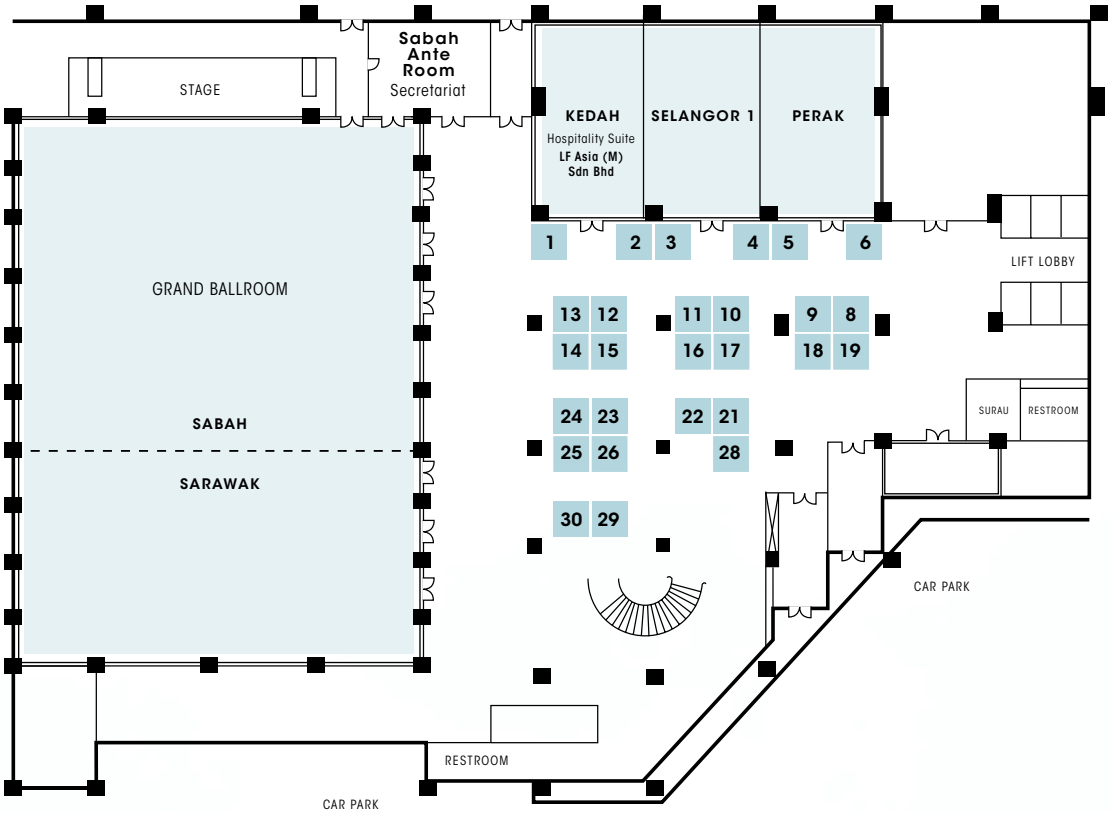

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CROHN'S DISEASE – NEW TREATMENT OPTIONS FOR A DIFFICULT DISEASE

Ng Siew Chien
Department of Medicine and Therapeutics, Chinese University of Hong Kong, Hong Kong

Crohn's disease can be severe, and is associated with complications including strictures and fistulas. Current treatment paradigm supports earlier treatment to achieve mucosal healing in selected patients. Although early combined immunosuppressive drugs were not more effective than conventional management for controlling Crohn's disease symptoms, they were associated with less adverse outcomes.

Commonly used biologics in Crohn's disease include anti-TNF-α agents (Infliximab, Adalimumab, Certolizumab). Approximately 30% of patients with Crohn's disease will not respond to induction therapy, and of those who initially respond, 50% will lose response within a year. Alternative therapeutics targeting other immune pathways are needed for patients refractory to conventional therapy. Novel therapeutic agents that are promising include selective anti-integrin therapy, anti-α4β7 antibody (MLN-002, vedolizumab) and the anti-cytokine antibody therapy directed to p40 subunit of IL-12 and IL-23 (Ustekinumab). In refractory Crohn's disease, among patients who had initially responded to induction therapy, ustekinumab maintenance therapy resulted in a higher rate of clinical remission and steroid-free remission when compared with placebo at week 22.

The optimal duration of treatment with biological agents for patients with Crohn's disease is not clear. Exit strategies may be necessary due to cost and long term complications of drugs, especially in Asian countries. Approximately 50% of patients who discontinued anti-TNF agents after combination therapy maintained remission 2 years later. The proportion in remission decreased with time. Severe disease activity, complicated disease course and poor prognostic factors, were associated with future relapse. Overall half or more of patients with IBD who cease therapy have a disease relapse. De-escalating treatment strategy should be mainly considered in patients with high risk of severe adverse events and low relapse risk (patients in deep remission) after drug withdrawal. This decision should be made for each individual based on patient preference, disease markers, consequences of relapse, safety, and cost.

CURRENT TREATMENT PARADIGM AND OUTCOME OF EARLY BIOLOGICS FOR ULCERATIVE COLITIS

John K Marshall
Gastroenterology Unit, Hamilton Health Sciences, Hamilton, Ontario, Canada

The clinical course of ulcerative colitis is heterogeneous, but risk factors such as disease extent and early requirement for corticosteroids can identify patients at increased risk for disease progression and colectomy. Aminosalicylates are an effective induction and maintenance therapy for mild-moderate disease. Corticosteroids are indicated when aminosalicylates fail, and are a first-line therapy for moderate-severe disease. However, every course of systemic corticosteroids should trigger a careful review of the maintenance strategy in order to avoid repeated exposure and steroid dependence. Anti-TNFα biologic therapy should be considered early for steroid-dependent and steroid refractory ulcerative colitis, and as a first line therapy for patients with severe or high-risk disease. The safety profile of anti-TNFα therapy is both manageable and favourable. With both intravenous and subcutaneous agents available, patient preference for route of administration can be considered. Better understanding of inter-individual variation in drug clearance can help to sustain remission through dose optimization in patients experiencing secondary loss of response. While colectomy can be appropriate for select patients with fulminant or refractory disease, its long-term outcomes are suboptimal and normal bowel function is not restored. Access to better medical therapy has reduced rates of colectomy for ulcerative colitis, and new agents on the horizon are likely to reduce these rates even further.

LECTURE 1

H. PYLORI AND PATHOPHYSIOLOGY OF GASTRODUODENAL AND OESOPHAGEAL DISEASE

Kenneth McColl

Institute of Cardiovascular and Medical Sciences, University of Glasgow, Glasgow, United Kingdom

H. pylori infection was first described 33 years ago. Since then, it has been found to play an important role in all the major upper GI diseases, which include peptic ulcer disease, gastric cancer and gastroesophageal reflux disease and its complications of Barrett's oesophagus and oesophageal adenocarcinoma.

H. pylori infection is the main cause of duodenal ulcer disease. In these subjects, the infection is association with a non-atrophic antral predominant gastritis. The ammonia produced by the organism's urease activity raises mucosal surface pH and this pairs the negative feedback control of gastrin release and acid secretion. Consequently, the infection results in increased duodenal acid load and duodenal ulceration.

H. pylori infection is also recognised to be the major acquired factor in the aetiology of non-cardia gastric cancer. In such subjects, the infection produces inflammation and atrophy of the entire gastric mucosa including the acid-secreting body region of the stomach. This hypochlorhydria and inflammation promotes carcinogenesis and explains the development of gastric cancer.

Recently, there has been interest in the strong negative association between *H. pylori* infection and oesophageal reflux disease and its malignant complications. It has been suggested that this might be related to the infection inducing atrophy and reducing acid secretion. If this is the mechanism, then this would need to involve the majority of those with the infection. In order to investigate this, we have recently examined gastric mucosa and gastric secretory function in the general population with and without *H. pylori* infection. We have found that the infection is associated with reduced acid secretion which is most marked close to the gastroesophageal junction, resulting in *H. pylori* infected subjects having little evidence of an acid pocket. We have also found that the *H. pylori* infected general population had a substantially reduced density of acid secreting parietal cells and pepsin secreting chief cells. These recent findings provide an explanation for the negative associations between the infection and reflux disease.

The falling incidence of *H. pylori* over recent years can explain the fall in DU and gastric cancer as well as the rise in GERD and its complications.

LUNCH SATELLITE SYMPOSIUM (ABBVIE)

SVR: WHAT IT MEANS IN REAL WORLD

Ashley Brown

St Mary's and Hammersmith Hospitals, London, United Kingdom

When the results of clinical trials using direct-acting antiviral drugs in chronic HCV were published just a few years ago, the results seemed nothing short of miraculous. But can these results – based on carefully selected patients treated in specialist centres – be replicated in the real-world?

This lecture will review data from recent congresses that reports on outcomes in routine clinical practice. What can we learn from these results and how will they help us shape future treatment programmes?

LECTURE 2

LONG TERM PPI - SHOULD WE BE CONCERNED?

Peter Katelaris

Gastroenterology and Liver Department, Concord Hospital, University of Sydney, Sydney, Australia

Proton pump inhibitors (PPIs) are among the most used drugs worldwide, with hundreds of millions of patient years of exposure over the last 25 years, and much clinical trial data. PPIs are considered safe and effective with a good risk-benefit profile when used for the appropriate indications of gastro-oesophageal reflux disease, NSAID ulcer healing, prophylaxis and treatment of upper gastrointestinal bleeding and as part of H. pylori eradication therapy. As with any drug, the risk-benefit changes when the drug is used inappropriately. For PPIs, commonly this includes the prolonged empirical use of PPIs for non-responsive and non-specific upper gut symptoms or as unnecessary prophylaxis in low risk patients.

Serious, idiosyncratic adverse effects (such as acute interstitial nephritis) are rare. Nonetheless, with drugs that are very widely prescribed even a very low relative risk may result in a substantial absolute number of such events.

Several putative adverse effects have been associated with PPI use, reported mostly from retrospective epidemiological studies. Such non-randomised, heterogeneous studies may suggest an association but do not prove causation. Reports include a small increased risk of infection (nosocomial and community acquired pneumonia, C.difficile infection and traveller's diarrhoea), adverse effects on bone health, micronutrient malabsorption, hypomagnesaemia, drug interactions, an increased risk of myocardial ischaemia, renal disease, microscopic colitis and more recently impaired cognitive function. The likelihood (odds ratio) of any risk appears to be modest in these studies, and associated usually with long term use and higher dosages. PPI monotherapy in the presence of H.pylori infection worsens gastritis scores and accelerates the development of gastric mucosal atrophy and intestinal metaplasia.

The guiding principle for PPI therapy remains to use these agents for appropriate indications, at the lowest effective dose for the shortest time necessary to ensure maximum benefit with minimised risk. Meanwhile, ongoing pharmacovigilance is paramount.

SYMPOSIUM 2 – FGID

NEW UNDERSTANDING OF VISCERAL HYPERSENSITIVITY IN FUNCTIONAL GASTROINTESTINAL DISORDERS (FGID)

Sutep Gonlachanvit

Gastrointestinal Motility Research Unit, Division of Gastroenterology, Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

Visceral hypersensitivity is a major pathogenesis mechanism of FGIDs. Abnormal brain-gut axis plays roles in the development of visceral hypersensitivity. Visceral hypersensitivity has been extensively studied in irritable bowel syndrome (IBS) and functional dyspepsia (FD). Several mechanisms have been recently demonstrated or proposed to be involved in the pathogenesis of visceral hypersensitivity in FGID including genetic, psychological or physical stress, change of intestinal permeability, altered gut microbiota, infection and low grade GI inflammation.

Visceral hypersensitivity leads to lower threshold for perception of gastrointestinal sensations or symptoms such as burning pain, cramping pain, bloating or urge to defecate in response to peripheral stimuli including change in gut wall tension, epithelial injury and chemical stimuli on afferent receptors of the gut mucosa. Most of gastrointestinal symptoms in FGID patients develop after ingestion of foods. Food ingestion can aggravate GI symptoms in FGID patients via mechanical and chemical stimulations on gut mucosal or gut wall receptors. Recently, it has been more research studies on the effect of foods on symptom development in FGID especially in IBS. Effects of FODMAP and gluten containing diets have gained more attention. Interaction between high FODMAP diet or increase intestinal gas production and visceral hypersensitivity on the development of GI symptoms in IBS has been reported. Understanding the interaction of visceral hypersensitivity and effects of food on the development of GI symptoms will help managing patients with FGODs.

REFLUX DISEASE NOT RESPONDING TO PPI

Kenneth McColl
Institute of Cardiovascular and Medical Sciences, University of Glasgow, Glasgow, United Kingdom

Failure of reflux symptoms to respond to PPI therapy is a major cause of referral to Gastroenterology clinics. Before labelling a patients as failure to respond to PPI therapy, it is important that you ensure that the patient has been taking the PPI in an adequate dose, for example, Omeprazole 40mg per day or 20mg twice per day, that they are taking the medication 30 minutes before food, that they are taking the medication regularly and that they appreciate that the medication has to be taken continuously as the treatment controls symptoms but does not cure the underlying disease.

If the patient has been taking the medication appropriately, then the cause of persisting symptoms is nearly always that the symptoms are not due to acid reflux. Many conditions produce reflux symptoms other than acid reflux. Some patients suffer from high volume reflux where they may wake up through the night choking and this condition does not respond well to PPI therapy and may require laparoscopic fundoplication if there is no other benefit from weight loss or other lifestyle measures. A variety of other conditions should also be considered in patients not responding to PPI therapy. These include cardiac pain, musculoskeletal pain, side effects of NSAIDs for other medication, candid oesophagitis, oesophageal motility disorders or eosinophilic oesophagitis.

The most common cause for reflux symptoms not responding to PPI therapy is that the patient suffers from functional heartburn. The symptoms are indistinguishable from acid-induced heartburn but there is no evidence of increased acid reflux and no association between any episodes of reflux and symptoms. As with other functional diseases, there is often an association with recent stressful life events, chronic fatigue syndrome, anxiety and depression and other functional diseases involving the GI tract, urinary tract or gynaecological problems. Diagnosis of functional heartburn should ideally be made on the history including poor response to PPI therapy and should not be a diagnosis based on exhaustive exclusion of other diseases. The treatment of functional heartburn consists predominantly of explanation and reassurance and may involve the management of psychosocial issues.

THERAPEUTIC CONSIDERATION IN INTESTINAL DYSBIOSIS

Justin Wu Che Yuan
Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong

Intestinal dysbiosis has been implicated in the pathophysiology of functional gastrointestinal disorder. There are numerous studies showing a significant difference in the gut microbiota between FGID patients and normal subjects. Alteration of specific intestinal bacterial populations has been linked to certain phenotypes of FGID. There is also mounting evidence suggesting a causal relationship between intestinal dysbiosis and functional gastrointestinal disorder, notably functional bowel disorders. The putative mechanisms include malabsorption, fermentation, immune dysregulation, motility dysfunction, visceral hypersensitivity, mood regulation and increased intestinal permeability, etc. Owing to these associations and biological plausibility, it has been postulated that modification of gut microbiota may have therapeutic implication on FGID. To date, most studies focus on the treatment of irritable bowel syndrome. Rifaximin, a poorly-absorbed, luminal-active antibiotic, has been approved for the treatment of bloating symptoms in IBS patients. However, the long-term effectiveness and safety, in particular the concern about induction of antibiotic resistance, remain to be confirmed. There are many small-scale trials supporting the use of probiotics in the treatment of IBS. However, the optimum species, route of administration and regimen have not been clearly defined. Prebiotics have also been studied for the treatment of IBS but the results are conflicting and the use may be limited by the potential side effects. Dietary modification, notably low FODMAPs meal, has been shown to be effective as a first-line treatment for IBS. Recent studies show that low FODMAP meal helps modify the gut microbiota, which leads to alleviation of visceral hypersensitivity and IBS symptoms. The recent advances in faecal microbiota transplantation (FMT) lead to the effective treatment of refractory Clostridium difficile infection through restoration of gut microbiota. However, its clinical application, practicalities and safety in the treatment of FGID is still poorly understood and further large-scale studies are needed.

HIGH RISK POPULATIONS: TO SCREEN OR NOT TO SCREEN?

Vincent Wong Wai Sun
Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Hong Kong

Non-alcoholic fatty liver disease (NAFLD) has become the most common chronic liver disease worldwide. The detection of NAFLD and its active form steatohepatitis (NASH) may identify patients for treatment. Patients with cirrhosis can also benefit from hepatocellular carcinoma (HCC) and varices surveillance. However, current guidelines have provided conflicting recommendations on screening. In this talk, I will use the Wilson and Jungner classic screening criteria to discuss whether screening should be recommended.

While NASH can progress to cirrhosis and HCC, it is important to remember that the majority of NAFLD patients run a benign clinical course and will never die from liver disease. With the development of non-invasive tests of fibrosis, it is possible to screen patients for NAFLD and fibrosis at the same setting and identify those who may need further evaluation. In a study of 1918 patients with type 2 diabetes, 73% and 18% were found to have NAFLD and advanced fibrosis by transient elastography, respectively, suggesting diabetes is a high risk group worthy of assessment.

Looking ahead, the follow-up action should be established before screening can be recommended. Lifestyle modification is an effective treatment for NASH but is difficult to implement and maintain. Besides, high risk groups require lifestyle modification anyway, and it is unclear if NAFLD screening adds to the management. Several drugs have been shown to reverse histological NASH in a minority of patients, and very few can reverse liver fibrosis; the impact on clinical outcomes remains uncertain. As yet, the effectiveness and cost-effectiveness of screening are not established.

But this is not the final answer. Further development in diagnostic tests and NASH treatment will likely make screening more effective. Meanwhile, the discovery of NASH biomarkers and studying the prognostic significance of non-invasive tests should be a research priority.

NAFLD PATIENTS WITH ADVANCED FIBROSIS - TREATMENT STRATEGY

Sombat Treeprasertsuk
Chulalongkorn University, Bangkok, Thailand

In Asian population, the reported prevalence of NAFLD was significantly high which varied from 12.4%-19.3% in the non-obese population, and 60.5% in obese subjects[1,2]. Recent study showed that presence of advanced fibrosis (F3-4) in NASH patients with was associated with the shorter survival than those without advanced fibrosis[3].The current strategies to improve liver inflammation and fibrosis include the combination of lifestyle modification and exercise with target of 7–10% weight loss of most lifestyle interventions[4]. The important issues in dietary changes are avoiding high-calorie diet for example reducing added sugar, avoiding sugar beverage or high fructose meal, reducing saturated or trans-fat or high cholesterol content as well as minimizing the processed food[5]. Exercise including both aerobic exercise and resistance training is an effective process to reduce liver fat[4].The achievement of NASH resolution by paired liver biopsy was about 25% in a year of follow-up[6]. Additionally, 90% of those NASH patients who achieved weight loss of at least 10% showed NASH resolution by histology whereas those NASH patients with 7-10% weight loss had reduced NAS score[6]. Recently, the noninvasive prediction model for NASH resolution which composed of 5 variables including weight loss (OR 2.75), type 2 DM(OR 0.04), ALT normalization(OR 9.84), age(OR 0.89), and a NAS≥5(OR 0.08)has been developed[7]. Bariatric surgery should be considered if indicated and it improved the overall mortality as well as reducing the liver inflammation and fibrosis[8]. Due to the multiple pathogenic mechanism, the development of new pharmacotherapies for NASH have some limitations for example the imperfect of animal model or the difficulty in surrogate markers for response of therapy. However, there was novel therapies for NASH with favorable outcomes[9]. Elafibranor is the promising agent which isan agonist of the peroxisome proliferator-activated receptor-α and peroxisome proliferator-activated receptor-δwith improvement of insulin sensitivity, glucose homeostasis and lipid metabolism[10].The GOLDEN study showed that Elafibranor 120 mg/day for 1 year had NASH resolution without fibrosis worsening in 20% of patients compared to 11% in placebo group[10]. The other promising future drugs in NASH are selective FXR agonists, metabolic regulators (e.g. FGF-19) and anti-inflammatory agents (e.g.CCR2, CCR5-antagonist; Cenicriviroc)[11,12]. Finally, to monitor disease severity, patients with NASH with advanced fibrosis should be monitored annually whereas those with cirrhosis should be monitored at 6 months intervals[4].

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HEPATITIS C - DAWN OF A NEW ERA IN TREATMENT

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Over the past five years we have seen a revolution in the treatment of Chronic Hepatitis C. Successive waves of new drugs offer more acceptable treatment regimens and dramatically improved outcomes. However they also throw up new challenges.

How can we ensure that those populations who would benefit from these drugs can access them and what impacts will this have on our service delivery? How can we ensure that nobody is left behind? What other obstacles lie between us and the laudable aim of eliminating HCV as a public health issue over the next decade?

TREATMENT TO ERADICATE HEPATITIS B- IS THERE LIGHT AT THE END OF THE TUNNEL?

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HBsAg seroclearance before the age of 50 years of age is associated with decreased incidence of hepatocellular carcinoma. Studies of spontaneous HBsAg seroclearance select special population with low viral activities. However HBsAg seroclearance is only achievable in 8-11% of patients with the current treatment agents. The pattern of HBsAg level decline during NA treatment is variable. In contrast, permanent suppression of HBV DNA levels to below PCR detectability is achieved in 98-100% of patients on long-term nucleoside analogues (NAs).

Hepatitis B virus (HBV) covalently closed circular (ccc) DNA, a minichromosome essential for HBV replication, is supposed to be resistant to NA treatment. In a pioneering study of 43 subjects with three liver biopsies on long-term NAs with continuous viral suppression for a median of 126 months, at the time of the third biopsy, serum HBV DNA levels were undetectable in all but one patient. Compared to baseline levels, there was reduction of HBsAg levels by 71.46%, ihHBV DNA levels by 99.84% and cccDNA levels by 99.89%, with 49% of patients having undetectable cccDNA. The median pregenomic RNA level, measured only in the third biopsy, was 0.021 copies per cell, with 40% of patients having undetectable pgRNA. Only one patient had undetectable HBsAg. With the low level of viral replication as indicated by the low cccDNA and pgRNA levels, it is likely that HBsAg is produced from integrated HBV DNA.

There are several agents currently being investigated. These agents can be classified into two main subgroups, direct acting antiviral agents and host-immune-stimulating agents. The former group includes HBV entry-to-hepatocyte inhibitors, short interfering RNA (siRNA), nucleocapsid assembly inhibitors. The latter group includes therapeutic vaccine and toll-like receptor agonists. These upcoming agents may further optimize HBV control. For clearance of integrated HBsAg, siRNA is the most likely option.

LECTURE 4

MANAGING COMPLICATIONS OF LIVER CIRRHOSIS AND PORTAL HYPERTENSION – VARICEAL BLEEDING AND HEPATORENAL SYNDROME

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In patients with decompensated cirrhosis, the development of variceal bleeding or hepatorenal syndrome (HRS) is often associated with poor outcome and high mortality rates without liver transplantation. Variceal bleeding occurs at a rate of approximately 5-15% per year in those with established cirrhosis, and treatment comprises of general supportive measures, pharmacological therapy, endoscopic therapy, and rescue therapy for those who have failed pharmacologic and endoscopic therapies.

For those with cirrhosis and ascites, the annual in incidence of hepatorenal syndrome (HRS) is an estimated 8%, with a prevalence rate ranging from 13% to 45%. The prognosis is generally poor, with survival usually measured in weeks and months for type 1 and 2 HRS respectively. The pathophysiology of HRS is largely an effect of peripheral vasodilatation, and therefore the primary medical therapy employs the use of vasoconstrictor therapy to improve renal perfusion. The use of albumin increases the effective arterial blood volume, further augmenting the effect of vasoconstrictor therapy. For those that relapse after stopping therapy, re-treatment is often effective, and may be required over a prolonged period as a bridge to liver transplantation.

The ultimate treatment for decompensated cirrhosis and HRS is liver transplantation, which significantly improves long-term survival. Therefore, timely referral to a transplant center is essential.

SYMPOSIUM 5 – HCC

SELECTION OF HCC PATIENTS FOR LIVER RESECTIONS

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Hepatocellular carcinoma (HCC) is a complex disease to manage because of its heterogenous nature as well as the concomitant presence of liver cirrhosis which can have a profound impact on the choice of therapeutic options. Surgery, either resection or transplantation, is currently the main therapeutic option with curative intent. Ablative methods are increasingly reaching results similar to surgery in selected tumour sizes in specific patients. The large majority of patients, however, are only amenable to palliative treatment of some sort due to the advanced nature of disease. Many staging systems have been proposed that hope to guide treatment options and the current most accepted one is the Barcelona Clinic Liver Cancer (BCLC) staging system. However, because of the heterogenous nature of disease and the complexities related to the associated cirrhosis and its complications, treatment choices and selection become more difficult and staging systems proposing guidelines may not be the best option to guide treatment. A more customised approach, relying on multidisciplinary board discussions and deliberations may be more appropriate to decide on treatment allocation. This presentation will discuss the current guidelines proposed by the staging systems and other considerations in patient selection for surgery beyond these guidelines.

THERAPEUTIC APPROACH TO LIVER CANCER- WHAT DO WE HAVE AND WHAT CAN WE DO FOR OUR PATIENTS?

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Hepatocellular carcinoma (HCC) is the sixth most common malignant disease and third most frequent cause of cancer death worldwide. HCC is prevalent in East Asia and sub-Saharan Africa, an endemic area of hepatitis B virus (HBV) and hepatitis C virus (HCV) infection. Currently, BCLC staging system is widely adopted by many countries, as well as EASL and AASLD. According to BCLC, in early stage of HCC (BCLC 0-A), either surgical resection, percutaneous ablation, or liver transplantation is a potential curative option and may provide 5-year survival rate up to 75 %. For patients not suitable for curative treatment and in the intermediate stage (BCLC B), transarterial chemoembolization (TACE) can provide locoregional tumor control to increase the survival. Sorafenib, a multikinase inhibitor is currently the standard of care for HCC patients in advanced stage of HCC (BCLC C). Several studies had tried to improve the recurrence-free survival for early stage of HCC or improve the progression-free survival of intermediate stage patients by adjuvant targeted therapy after curative resection/ablation or TACE. However, the survival benefit was not achieved, even though combination TACE with sorafenib is documented to be safe and tolerable. In patients with advanced HCC, many clinical trials including brivanib, tarceva, everolimus and other targeted therapy were not superior to sorafenib. In patients who failed by sorafenib, a recent trial shows an exciting result by regorafenib. Immunotherapy is a rising star in the treatment of cancer and has been documented to prolong survival in melanoma, lung cancer and colorectal cancer.The preliminary report in HCC study also shows promising.A novel transarterial treatment modality,Yttrium-90 TARE, might apply in patients with portal vein tumor thrombus to downstage the tumor.All HCC patients should be treated individualize and multi-modality approach is usually required to maximize the treatment outcome.

LIVER TRANSPLANT FOR HEPATOCELLULAR CARCINOMA – IS THIS THE BEST OPTION?

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Hepatocellular carcinoma (HCC) is currently the third most common cause of cancer-related death in the Asia-Pacific region. Liver transplantation (LT) offers the highest potential for cure when compared to all other available treatment modalities. In addition, LT offers a cure for those with established cirrhosis, and removes any future potential development of metachronous HCCs. On these facts alone, LT would appear to be the best treatment option for HCC patients. In fact, LT is often the only curative option available, particularly for those with advanced cirrhosis not amenable to surgical resection or locoregional therapies. For these patients, LT is the best option if the HCC is within the adopted criteria. However, despite the use of well-tested selection criteria, recurrence of HCC can occur with significant reduction in long-term survival. Other parameters beyond tumour size and number, including AFP and specific characteristics on imaging, may also have a role in determining which patients will benefit most from LT. Furthermore, organ shortage remains the single most important restriction in most transplant programs, and eligible wait-listed patients may still succumb to disease progression, even with the use of bridging therapy. Unfortunately, current existing eligibility criteria for LT in HCC patients are not individualized, and may in fact not identify the best patients, as some within criteria may display other characteristic tumor features associated with poor outcomes after transplantation. On the other hand, ineligible patients beyond the adopted criteria may not have compromised long-term outcome upon careful selection.

BEST PAPER AWARD PRESENTATIONS

THE DEMOGRAPHIC, CLINICAL FEATURES, RESPONSE TO TREATMENT AND OUTCOME OF 109 AUTOIMMUNE HEPATITIS (AIH) PATIENTS IN MULTI-ETHNIC MALAYSIA SINCE 2002

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OBJECTIVES

AIH occur in all ages worldwide with variations due to genetics and environmental factors. There is a paucity of AIH data in Malaysia. We aim to study the sociodemographic and clinical presentations, and outcome in AIH patients under our follow-up.

METHODS

Retrospective analysis of a prospectively maintained AIH database according to Simplified Diagnostic or Revised International Autoimmune Hepatitis Group Criteria.

RESULTS

109 patients were identified (86 female; 79 Malay, 21 Chinese, 7 Indians, one Bumiputera Sarawak and two others; median age at presentation 47 years (range 11-72). Type 1 AIH (n=82, 75%), type 2 AIH (n=2, 2%), autoimmune markers negative (n=14, 13%) and overlap syndrome (n=11, 10%). The mode of presentations were acute hepatitis (n=47, 43%), chronic hepatitis (n= 22, 20%), compensated cirrhosis (n=9, 8%), decompensated cirrhosis (n=22, 20%), acute-on –chronic liver failure (n=2, 2%) and acute liver failure (n=7, 7%). The median MELD score at presentation 19 (range 6-43). Twenty three patients had preceding history of consuming traditional and complementary medicines. Pretreatment liver biopsies were performed in 98 patients. A total of 28 patients (26%) were diagnosed with concurrent extrahepatic disorders with thyroid disease the most common (n=11, 10%). Prednisolone monotherapy was the predominant immunosuppressive agent used at initiation (n=85, 78 %). The maintenance immunosuppression was prednisolone plus azathioprine (n=51, 47%), prednisolone with MMF (n=3, 3%), prednisolone monotherapy (n=18, 17%) and azathioprine monotherapy (n=21, 19%), 12 patients were not on maintenance therapy and 4 underwent liver transplantation. After a median follow-up of 47 months (range 1-160), 53(49%) showed a complete biochemical response, 34(31%) partial, 1(1%) treatment failures, 17(16%) died (10 non-liver related, 1 hepatocellular carcinoma), and 4 defaulted.

CONCLUSIONS

AIH afflicts predominantly females of all ages, type 1 AIH being most common. It has variable mode of clinical presentations, their recognition and prompt diagnosis will ensure good outcome.

THE ROLE OF GUT DYSBIOSIS AND PROBIOTICS IN PERSISTENT ABDOMINAL PAIN FOLLOWING A MAJOR FLOOD DISASTER

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INTRODUCTION

On December of 2014, parts of northern Malaysia experienced a massive river-flood which has contributed to significant morbidity. Chronic abdominal pain has been reported post-flood, although exact mechanisms remain unclear.

WE AIMED TO

(1) determine the association of small intestinal bacterial overgrowth (SIBO) and chronic abdominal pain;

(2) describe the gut microbial taxa of patients with chronic abdominal pain; and

(3) evaluate the effect of probiotics on symptom improvement.

METHODS

Baseline demographics, glucose hydrogen breath test and stool analysis for 16S rRNA pyrosequencing were performed in consented flood victims. Participants with chronic abdominal pain were identified and a group of asymptomatic participants from the same population were included as controls. The probiotic, Bifidobacterium infantis M63 (109 colony forming units) 1g sachet daily was given for 3 months in the abdominal pain group. Pre- and post-treatment evaluations included SF-36 quality of life assessment and a subjective symptom assessment score. Primary outcome was improvement in SF-36 and symptom scores. A p-value < 0.05 was considered significant.

RESULTS

A total of 211 flood victims were recruited and 38% experienced chronic abdominal pain. SIBO was present in 12.6% (N=17) and it was significantly associated with chronic abdominal pain ($p=0.02$). The most common phyla were Bacteroidetes (37%), Firmicutes (25%) and Proteobacterial (8%). More Fusobacteria was seen in participants with abdominal pain ($P=0.003$) while Firmicutes was higher in those with SIBO ($p=0.03$). Twenty participants with chronic abdominal pain were given probiotics vs 32 controls. Mental well-being and subjective symptom score were significantly better post-probiotic ($p=0.002$, $p<0.001$, respectively). More Bacteroidetes (57%) and lower Firmicutes (24%) were noted post-probiotic compared to controls ($p=0.02$ & $p=0.008$).

CONCLUSION

Chronic abdominal pain is associated with presence of SIBO. Fusobacteria is more common with abdominal pain and Firmicutes in SIBO. Probiotic supplementation improves patient's abdominal pain and mental well-being.

THE APPLICATION OF SERUM BIOMARKERS TO DETECT PRE-MALIGNANT LESIONS IN GASTRIC CORPUS

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OBJECTIVE

Gastric adenocarcinoma is often diagnosed at advanced stage, leading to cancer death. Corpus-predominant atrophic gastritis increases the risk of gastric cancer. We aim to investigate the utility of serum biomarkers to diagnose chronic atrophic gastritis (CAG) and intestinal metaplasia (IM) and determine the sensitivity and specificity of serum pepsinogen I (PGI), pepsinogen II (PGII), ratio of PGI to PGII (PG I/II) and gastrin-17 (G-17) in detecting these lesions.

METHODOLOGY

We performed a cross sectional observational study involving patients who underwent gastroscopy for dyspepsia in our unit. Endoscopic CAG is graded based on Kimura-Takemoto classification and gastric biopsies were analyzed using updated Sydney system. Serum PGI, PGII, G-17 and H. pylori antibody levels were measured by enzyme-linked immunosorbent assay.

RESULTS

A total of 72 patients with mean age of 56.2 years (±16.2) were recruited. The median level of PGI, PGII, PG I/II ratio and G-17 for all subjects were 129.9µg/L, 10.3µg/L, 14.7 and 4.4pmol/L respectively. Subjects with corpus CAG/IM had significantly lower PG I/II ratio (7.2, $p < 0.001$) compared to the control group (PG I/II=15.7). There was no significant difference in serum G-17 level between antral CAG/IM group and non-CAG group. Histological CAG and IM correlated well with serum PG I/II ratio ($r = -0.417$, $p < 0.001$). The cut off value of PG I/II ratio of ≤ 10.0 exhibit high sensitivity(83.3%), specificity(77.9%) and area under the ROC curve (AUC) of 0.902 in detecting corpus CAG/IM. However, at PG I/II ratio of ≤ 3.0, the sensitivity was very low. Serum PG I, PGII and G-17 level have low sensitivity in detecting CAG/IM.

DISCUSSIONS AND CONCLUSION

Serum PG I/II ratio could potentially be used as an outpatient and non-invasive method for detecting pre-malignant gastric lesions, in particular chronic atrophic gastritis and intestinal metaplasia in gastric corpus.

KEYWORDS

gastritis, atrophic; metaplasia; pepsinogens; gastrin-17

ACCURACY OF STANDARDIZED PROTOCOL IN DIFFERENTIATING CROHN'S DISEASE FROM INTESTINAL TUBERCULOSIS IN A SOUTH-EAST ASIAN POPULATION

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INTRODUCTION

The differentiation between intestinal tuberculosis (ITB) from Crohn's disease (CD) remains a challenge, particularly in areas where TB is highly prevalent. Previous studies have identified features that favour one diagnosis over the other.

AIMS

To determine the accuracy of a standardized protocol in the initial diagnosis of CD versus ITB.

METHODOLOGY

All patients with suspected ITB or CD were prospectively recruited. A standardized protocol was applied and the diagnosis of probable ITB or CD was made accordingly. The protocol consists of; history and examination, ilecolonoscopy with biopsies, TB cultures+/-PCR, Mantoux/IGRA test, CT scan chest and abdomen. The diagnosis of probable TB was made based on the at least one of the following criteria: close contact with TB, AAFB on biopsy, positive TB PCR/cultures, multiple granulomas and/or large granulomas on histology, caseating and/or large intra-abdominal nodes, ascites, abnormal lung findings on CT and positive Mantoux/IRGA. All other patients were diagnosed as probable CD. Patients were treated either with anti-tuberculous therapy or steroids. Reassessment was then carried out clinically, biochemically and endoscopically at 8 weeks. In patients with suboptimal response, the diagnosis was changed and the treatment was changed accordingly.

RESULTS

107 patients were recruited. M:F 60:47;median age 28(range 1,75);Malay 20(18.7%) , Chinese 36(33.6%), Indians 47(43.9%), Other 4(3.8%). The final diagnoses were as follows; 9 (8.4%) patients had ITB, 97(90.7%) had CD, one (0.9%) had a colonic lymphoma. 1(11.1%) out of 9 patients with ITB was initially treated as CD. 7(7.2%) out of 97 patients with CD were initially treated as TB. 105(98.1%) out of the 107 patients received the correct treatment by 12 weeks from initial workup. The initial overall accuracy for the protocol was 90.6%.

CONCLUSION

In our population, most patients had CD rather than ITB. The standardized protocol had a high accuracy in differentiating CD from ITB.

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DIABETES, MALE GENDER AND INCREASING AGE: SIGNIFICANT RISK FACTORS IN DEVELOPING GALLBLADDER EMPYEMA

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BACKGROUND

Gallbladder empyemas (GE) are dreaded conditions complicating cholelithiasis. They pose a diagnostic and treatment challenge to clinicians. Although published work on this condition is still lacking, GEs are known to cause significant morbidity and mortality particularly when not anticipated.

AIM

To identify the association between diabetes, male gender, patients' age and GE.

MATERIAL AND METHODS

This was an analytical cross-sectional study involving patients who underwent cholecystectomy during a 2-year period. 220 consecutive patients' records were analysed. The diagnosis of GE was confirmed intra-operatively and also histopathologically. Data were collected from operation theatre database and hospital medical records. Chi square and t test were performed using SPSS statistical software.

RESULTS

A total of 220 patients underwent cholecystectomy of which 26 patients had GE. The mean age of patients who was diagnosed with GE was 58.1 ± 10.7 years which was significantly older ($p<0.05$) than patients with other gallstone diseases. Male patients were significantly associated with GE (odds ratio [OR], 2.8; $p<0.05$) compared to their female counterparts. Patients with diabetes, a disease affecting 20% of our sample population were found to be more likely to develop GE (OR, 7.8; $p<0.05$) compared to those without this disease.

CONCLUSION

This study has concluded that increasing age, male gender and diabetes were significantly associated with the development of GE.

UTILIZATION OF GASTROPROTECTIVE STRATEGIES IN PATIENTS ON REGULAR NONSTEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS) - A SINGLE-CENTRE STUDY

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BACKGROUND & AIM

Gastroprotective strategies are recommended to reduce the risk of gastrointestinal (GI) adverse effects in at-risk patients on regular nonsteroidal anti-inflammatory drugs (NSAIDs). However, these recommendations have not been adequately translated into clinical practice. This study aimed to investigate the utilization of gastroprotective strategies in routine clinical practice.

METHOD

A cross-sectional study was conducted in the Outpatient Pharmacy of a large, tertiary institution in Malaysia. All patients who met the inclusion criteria for regular NSAID use were recruited and interviewed using a questionnaire.

RESULTS

The mean (standard deviation) age of the 409 participants recruited was 52.3(14.6) years, with 60.6% female. Of these participants, 60.6% were naïve and 39.4% were repeated NSAID users, for the following main indications: musculoskeletal pain 48.4%, osteoarthritis 13.9% and vertebro-degenerative disease 9.5%. The types of NSAIDs used were: 59.2% non-selective NSAIDs alone, 39.8% cyclo-oxygenase (COX)-2 inhibitors alone and 1% combination of NSAIDs. At least one GI risk factor was identified in 340 participants (83.1%), of whom only 30% received appropriate gastroprotection. The most common risk factors were the use of high-dose NSAIDs (69.2%), followed by age of participants ≥ 65 years (22%) and concomitant use of low-dose aspirin (11.7%). Logistic regression analysis revealed age ≥ 65 years [OR 1.89, 95% CI=1.15-3.09] as the main predictive factor for the prescription of gastroprotection by clinicians. Gastroprotective strategies were underutilized in 67.1% of at-risk participants and over-utilized in 59.4% of those without risk factors. Co-prescription of histamine-2 receptor antagonists at lower than recommended doses constituted 59% of the inappropriate gastroprotection strategies.

CONCLUSION

Utilization of gastroprotection strategies in patients on regular NSAIDs was unsatisfactory in this study. Further measures need to be implemented to improve the safe prescribing of regular NSAIDs.

14-DAY HIGH-DOSE DUAL THERAPY VERSUS 14-DAY CLARITHROMYCIN BASED STANDARD TRIPLE THERAPY: A PROMISING ALTERNATIVE THERAPY?

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BACKGROUND & AIMS

The efficacy of treatment of Helicobacter pylori infection has decreased steadily because of increasing resistance to clarithromycin. Our aim of this study is to examine the efficacy and tolerability of high-dose dual therapy (HDDT) versus clarithromycin based standard triple therapy (STT) as first-line eradication therapy.

METHODS

Consecutive treatment naïve participants with a positive rapid urease test during an outpatient upper endoscopy were included. All participants were randomly assigned to STT group given rabeprazole (Pariet) 20 mg b.i.d., amoxicillin (Ospamox) 1 g b.i.d. and clarithromycin (Klacid) 500 mg b.i.d. for 14 days and HDDT group given rabeprazole (Pariet) 20 mg q.i.d, amoxicillin (Ospamox) 1g q.i.d for 14 days. Successful eradication was defined by negative C13-urea breath test at least 4 weeks after the completion of therapy.

RESULTS

As an interim-analysis, a total of 30 patients were recruited. In the intention-to-treat and per-protocol analysis, H. pylori was eradicated in 100% of patients in the STT arm, (15/15) (95% CI: 79.61%–100 and 93.3% (14/15) (95% CI: 70.19%–98.81%) in the HDDT arm. There were no significant differences between adverse events in both groups.

CONCLUSIONS

14-day High Dose Dual Therapy is a promising alternative treatment to 14-day clarithromycin based triple therapy. We await analysis on a larger sample population to confirm our preliminary findings described here.

CHARACTERISTIC OF RED CELL INDICES ASSOCIATED WITH IRON DEFICIENCY ANAEMIA (IDA) AND ENDOSCOPIC FINDINGS

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OBJECTIVE

To study the relation of red cell indices in IDA and endoscopic findings

METHODOLOGY

We have retrospectively reviewed the patients who had undergone oesophagealgastroduodenoscopy (OGDS) and/or colonoscopy for iron deficiency anaemia from 1st January 2015 to 31st December 2015 from Malaysia Gastrointestinal Endoscopy Registry. A total number of 161 patients' endoscopy reports were reviewed. There were 47 patients confirmed to have iron deficiency anaemia with serum ferritin<30ng/L. Overall, structural lesions were found in 62% of the patients. The structural lesions include oesophagitis (12.5%), benign gastric ulcer (6.3%), gastric erosion (6.3%), benign duodenal ulcer (14.6%), oesophageal varices and portal hypertensive gastropathy (2.1%), gastric tumor (4.2%), colon carcinoma (10.2%) and colon ulcer (2.1%).

RESULTS

Of note, MCH<20 pg and MCV<60 fl have high positive likelihood ratio (14.00 and 7.35 respectively) for Serum Ferritin<30 ng/dL.

Indices	Sensitivity	Specificity	LR+	LR-	Assoc. structural lesion (p value)	Assoc. Colon carcinoma (p value)
MCV<60	25	96.6	7.35	0.78	0.035	<0.01
MCV<78	87.5	67.2	2.67	0.19	0.092	0.008
MCH<20	58.3	95.8	14	0.44	0.037	0.031
MCH<27	91.7	53.8	1.98	0.15	0.353	0.304

MCV, Mean corpuscular Volume; MCH Mean cosrpuscular haemoglobin, LR+ Positive likelihood ratio, LR- Negative Likelihood ratio; Assoc. Associated with, shading: Significant value: Positive likelihood ratio >5 or p value <0.05

Patients who had MCV<60 and MCH <20 are significantly associated with combined structural lesion (including oesophagitis, gastrroduodenal ulcer or erosions, oesophageal varices, portal hypertensive gastropathy, gastric tumor, colon carcinoma, or colon ulcer.) and colon carcinoma.

CONCLUSION

MCH<20 pg and MCV<60 fl may be a useful surrogate indices for iron deficiency anaemia. It was more likely to find a structural lesion in this group of patients.

LIVER ABSCESS IN MALAYSIA: WHAT HAS CHANGED?

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OBJECTIVES

The common types of liver abscesses seen are amoebic and pyogenic abscess. The prevalence of amoebic liver abscess has been closely associated with social economy status of a country. We set out to review liver abscesses that were diagnosed over a period of 2 years in University Malaya Medical Centre (UMMC) Kuala Lumpur.

PATIENTS AND METHODS

Patients that presented to UMMC from August 2014 to May 2016 that were diagnosed to have liver abscesses were identified and their records traced. Demographic characteristics, presenting histories, radio-imaging details, blood investigations and culture and sensitivity were captured.

RESULTS

Thirty five liver abscess cases were identified. 42.9% were Chinese compared to Malays (28.6%) and Indians (20%). The male to female ratio was 2:1. Nineteen cases were found to be pyogenic in origin, thirteen indeterminate and one of amoebiasis, tuberculous and penicilliosis each. 18/35 (51.4%) presented with classical symptoms of RUQ pain and fever. 17/35 (48.6%) were incidentally detected while being investigated for systemic illness. 24/35 (68.6%) of abscesses were confined at right lobe of the liver and only 4/35 (11.4%) were found to be solitary lesions. The organisms isolated from blood or pus cultures were 9/35 (25.7%) Klebsiella pneumoniae, 6/35 (17.1%) Escherichia Coli (non ESBL form) , 2/35 (5.7%) Enterococcus faecium , 1/35 (2.8%) Pantoea species and 2/35 (5.7%) Burkholderia pseudomallei, 1/35 (2.8%)Mycobacterium Tuberculosis ,1/35 (2.8%) penicillium marfennei while entamoeba histolytica was only successfully identified in a single case.

CONCLUSION

Majority of patient presented with the classical symptoms of RUQ pain and fever. Pyogenic liver abscess has taken over to be the predominant cause of liver abscess in our case series. We await more patients to substantiate our initial findings reported here.

A STUDY ON TRANSMISSION OF HEPATITIS B VIRUS INFECTION AMONG CHILDREN OF HEPATITIS B SURFACE ANTIGEN POSITIVE MOTHERS FROM A TERTIARY HOSPITAL IN MALAYSIA

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OBJECTIVE

Routine hepatitis B vaccination and the administration of hepatitis B immunoglobulin (HBIG) to infants born to HBsAg-positive mothers have been in effect in Malaysia since 1989. The objective is to study the transmission of hepatitis B virus (HBV) infection among children of hepatitis B surface antigen (HBsAg) positive mothers in Malaysia.

METHODOLOGY

This is a cross-sectional study of all the children of HBsAg-positive mothers who delivered at the University of Malaya Medical Centre between 1993 and 2000.

RESULTS

A total of 60 HBsAg-positive mothers and their 154 children participated in the study. HBsAg was detected in four children (2.6%) while anti-HBc IgG was detected in seventeen children (11.0%). The mother's age at childbirth was significantly lower in the children with detectable HBsAg (22.5 ± 6.1 years vs. 29.7 ± 4.5 years, p = 0.043) and anti-HBc IgG (26.6 ± 6.1 years vs. 30.0 ± 4.3 years, p = 0.004). Children born in the 1980s were significantly more likely to have detectable HBsAg (18.8% vs. 0.7%, p = 0.004) and anti-HBc IgG (37.5% vs. 8.0%, p = 0.000) compared with those born later. All the children with detectable HBsAg were born via spontaneous vaginal delivery, and HBIG was either not given or the administration status was unknown. The majority of mothers with chronic HBV infection (70.4%) were not under any regular follow-up for their chronic HBV infection and the main reason was the lack of awareness of the need to do so (47.4%).

DISCUSSION AND CONCLUSION

Transmission of HBV infection among children of HBsAg-positive mothers in Malaysia is low. However, attention needs to be given to the high rate of HBsAg-positive mothers who are not on any regular follow-up for their chronic HBV infection and the lack of awareness among them of the need to do so.

HEPATITIS C AND HIV CO-INFECTION TREATMENT OUTCOMES IN HOSPITAL SULTANAH BAHIYAH, ALOR SETAR, KEDAH

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OBJECTIVE

Human immunodeficiency and hepatitis C virus coinfection (HIV/HCV) poses a challenging treatment avenue where a careful selection of patients would aid in the improvement of success rate. We report our treatment outcomes from Hospital Sultanah Bahiyah, Alor Setar.

METHODOLOGY

A retrospective analysis of our success rate in treatment of HIV/HCV coinfection with pegylated interferon 2a/2b and ribavirin (PEG-IFN/RBV) from the year 2007 to 2016.

RESULTS

Sixteen HIV/HCV coinfected patients underwent PEG-IFN/RBV therapy from our study duration of which the mean age (standard deviation, SD) was of 41.1 years (7.23). All except three were males. We had eleven patients with genotype 3 (GT3) while five were genotype 1 (GT1). The mean (SD) CD4 count was 482.9 cells/μl (175.57). The overall sustained virological response (SVR) rates were 75% with the bulk coming from GT3 compared to GT1 (81.8% vs. 60%; p=0.547). All except two of our patients were treated with antiretroviral therapy (ART). Our treatment completion rate was 75% with the remaining 25% dropping out due to adverse events and treatment default. The SVR rates for patients with low viral load (<400,000 IU/mL) and high viral load (>400,000 IU/mL) were 100% and 33.3% respectively (p=0.516).

CONCLUSION

Though registered trials show a generally more responsive treatment towards PEG-IFN/RBV for GT 3 and lower viral load, ours was not statistically significant. Treatment outcomes were notably comparable suggesting that treatment should be offered to all when there are no contraindications. A close-knitted healthcare delivery system with multidisciplinary effort is recommended pretreatment.

CASE REPORT - SYNCHRONOUS CMV INFECTION IN A NEWLY DIAGNOSED ULCERATIVE COLITIS PATIENT

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Gastrointestinal infections with cytomegalovirus (CMV), especially colitis, are usually found in immunocompromised patients and rarely affect immunocompetent subjects.

We report the case of a 61 year old Punjabi female patient who presented with six months history of bloody diarrhea associated with mild abdominal discomfort. She denied of underlying chronic medical illness; she neither took steroid nor immunosuppressant. Biochemistry showed normochromic normocytic anemia with Haemoglobin level 8.8g/dL, reactive thrombocytosis; and elevated C-reactive protein. Colonoscopy showed proctitis. The histopathology examination reported as Ulcerative Colitis (presence of crypt abscesses, cryptitis, crypt distortion, lymphoplasmacytosis, and neutrophil infiltration) with CMV infection (nuclear positivities of CMV immunostain), otherwise, no granuloma/dysplasia/malignancy noted. After combined anti-inflammatory (both oral and suppository 5-ASA with intravenous hydrocortisone initially followed by tapering dose of oral prednisolone) and two weeks of intraveous ganciclovir, she recovered well.

Few literatures mentioned the significant prevalence of CMV infection in IBD. CMV reactivation is frequent in severe or steroid-resistant UC. It is not known whether the virus exacerbates the disease or simply appears as a bystander1. Specific endoscopic features have not been described in active UC and CMV infection. Most authors recommend the use of antivirals in steroid-refractory UC flare-up and CMV positive patients2. Treatment can significantly decrease the mortality rate and need for surgery.

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TREATMENT RESPONSE OF PEG-INTERFERON ALPHA PLUS RIBAVIRIN THERAPY IN PATIENTS WITH HEPATITIS C IN SERDANG HOSPITAL

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OBJECTIVES

To evaluate the treatment response of Peg-Interferon Alpha plus Ribavirin therapy in patients with Hepatitis C in Serdang Hospital.

METHODS

The demographic data (gender, age, ethnicity), viral genotype, HCV viral load, ultrasound finding of liver cirrhosis, and incidence of sustained virological response (SVR) were analyzed in 38 patients with Hepatitis C treated with Peg-interferon Alpha plus Ribavirin therapy from 2007-2015.

RESULTS

From 2007-2015, 38 out of the 172 patients with Hepatitis C in Serdang Hospital had completed Peg-Interferon Alpha plus Ribavirin therapy, with mean age 50.44 years old, and male 29/38 (76.32%), female 9/38 (23.68%). 65.79% of them were Malay, followed by Chinese (31.58 %), and Indian (2.63 %). Majority of the patients were of Genotype 3 (68.42%, n=26), with Genotype 1 28.95% (n=11), and Genotype 2 2.63% (n=1). 8 of them had radiological evidence of liver cirrhosis. SVR achieved: 72.73 % (8/11) in HCV Genotype 1, 100% (1/1) in HCV Genotype 2, and 76.92 % (20/26) in HCV Genotype 3.

CONCLUSION

We conclude that Peg-Interferon Alpha plus Ribavirin was useful in the treatment of Hepatitis C. SVR achieved in HCV Genotype 1 and 3 were consistent with the current data available.

DISCUSSION

The number of patients receiving antiviral treatment here is small, mainly due to the treatment cost.

OVERUSE / INAPPROPRIATE USE OF ACID SUPPRESSION THERAPY (AST) AS GASTROINTESTINAL PROPHYLAXIS IN GENERAL MEDICAL WARDS IN A MALAYSIAN DISTRICT SPECIALIST HOSPITAL

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OBJECTIVE

The primary objective is to determine the percentage of overuse/ inappropriate use of Acid Suppression Therapy (AST) as gastrointestinal prophylaxis and upon discharged in general medical wards in a district specialist hospital. To calculate the drug cost savings from preventable inappropriate usage of AST.

METHODS

Retrospective study. Total of 152 patients were recruited. Data collection from case notes of patients that were prescribed with AST (proton pump inhibitors or H2 antagonist) in general medical wards in Hospital Kulim in February 2016. Appropriateness of AST as gastrointestinal prophylaxis was determined based on best available guidelines from American Society of Health-System Pharmacists (ASHP) on stress ulcer prophylaxis (SUP) and European Society of Cardiology (ESC) recommendation on dual anti-platelet therapy (DAPT).

RESULTS

Out of 152 patients, 102 patients (67.1%) were prescribed AST for prophylaxis. More than half did not have appropriate indication for AST prophylaxis (55.9%, n=57) with 32 patients (56.1%) among them were discharged home with AST unnecessarily. Total cost savings from these amounts to RM521/month and projected to be at least RM6252/year. For inappropriate prescribing, sepsis syndrome (14.6% for SUP) and age≥65 (37.5% for DAPT) were identified as the most common relative indication.

DISCUSSION

Literatures internationally reported 33 to 65% inappropriate prescribing of AST, whereby our setting also is as high at 55.4%. This likely due to lack of guidelines and awareness on AST use in non-critically ill patients. This is alarming as more complications are reported with long-term AST use.

CONCLUSION

Overuse of AST in non-critically ill patients as inpatient and upon discharged is confirmed in this study. This can potentially lead to many adverse events besides wastage of resources. Awareness and education with protocol regarding proper AST prescribing should be provided to clinician.

CASE REPORT: UNEXPECTED FINDING OF ASCARIASIS DURING SURVEILLANCE OESOPHAGOGASTRODUODENOSCOPY (OGDS)

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INTRODUCTION

Ascariasis is one of the most common nematode infections in the world with Ascaris lumbricoides as the main agent responsible for human ascariasis. Human are infected by ingesting contaminated food with Ascaris eggs, especially in fresh vegetables and usually due to poor sanitation and hygiene. Most patients are asymptomatic but some may be complicated with intestinal obstruction, hepatobiliary or pancreatic ascariasis.

CASE REPORT

We report a case of a middle age gentleman, with underlying diabetes mellitus, who is asymptomatic, came for a surveillance oesophagogastroduedenoscopy (OGDS) after an event of upper gastrointestinal bleed (UGIB) secondary to peptic ulcer disease (multiple ulcers over stomach and duodenum) with helicobacter pylori infections 2 months ago. His hemoglobin level was 11.5 g/dl and eosinophil count was 0.40 10^3/uL.

RESULTS

Incidentally a roundworm was found in second part of the duodenum (D2). Otherwise the OGDS was normal. Patient was treated with Tablet Albendazole 400mg stat dose.

DISCUSSION

To be able to diagnose round worm on OGDS is rare as intestinal ascarids usually resides in jejunum and ileum.

CONCLUSION

Ascariasis can be diagnosed with OGDS unexpectedly. Therefore we need to be caution and not to miss the parasite whenever performing OGDS especially on patients with risk factors.

A STUDY OF NON-ALCOHOLIC FATTY LIVER DISEASE USING TRANSIENT ELASTOGRAPHY AND CAROTID INTIMA MEDIA THICKNESS USING ULTRASONOGRAPHY IN A MIDDLE-AGED MALAYSIAN COHORT

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OBJECTIVES

There has been no study on the association of NAFLD evaluated by using Transient Elastography (TE) and carotid intima media thickness (CIMT). The primary objective of this study was to determine the prevalence of NAFLD, advanced fibrosis and increased CIMT in a Malaysian population. The secondary objective was to assess for association between NAFLD/advanced fibrosis and increased CIMT.

METHODOLOGY

This was a cross-sectional study conducted in a government hospital in Kuala Lumpur from August 2015 until January 2016. The study participants consisted of the armed forces personnel (active and retiree) and their immediate family members. Demographic, clinical, anthropometric and biochemical measurements were recorded. Participants with significant alcohol intake and other causes of chronic liver disease were excluded. TE was performed using FibroScan® 502 Touch with M probe. NAFLD was present if Controlled Attenuation Parameter (CAP) ≥263dB/m while advanced fibrosis was present if Liver Stiffness Measurement ≥8kPa. Increased CIMT was defined as CIMT ≥0.8mm on carotid ultrasonography.

RESULTS

Data for 251 participants were analyzed (mean age 47.1±12.4 years, male 74.1%). 57.4% of the participants had NAFLD, 17.5% had advanced fibrosis, and 29.0% had increased CIMT. The independent factors associated with NAFLD were greater waist circumference ($p<0.001$) and elevated AST level ($p=0.035$). The independent factors associated with advanced fibrosis were male gender ($p=0.018$) and elevated AST level ($p=0.015$). Independent factors associated with increased CIMT were older age ($p<0.001$), diabetes mellitus ($p=0.020$), hypertension ($p=0.019$) and elevated LDL level ($p=0.025$). NAFLD subjects tend to have increased CIMT (34.5% vs. 19.1%, $p=0.063$). Advanced fibrosis was not associated with increased CIMT (30.4% vs. 28.7%, $p=0.868$).

CONCLUSION

NAFLD, advanced fibrosis and increased CIMT was found to be common in a middle-aged Malaysian population. NAFLD, but not advanced fibrosis tended to be associated with increased CIMT.

TELEMEDICINE FOR GASTROINTESTINAL ENDOSCOPY – THE ENDOSCOPIC CLUB E-CONFERENCE IN THE ASIA PACIFIC REGION

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OBJECTIVES

Regional gastrointestinal endoscopy-related teleconferences were traditionally organized by the medical working group (hosted by Kyushu University, Japan) of Asia-Pacific Advanced Network. Endoscopic Club E-conference(ECE) was set up in May 2014 to cater for the increased demand of such activities. This study described the running of ECE meeting, examined the group dynamics, outlined their feedback and analyzed factors affecting participation enthusiasm. It is hoped that the findings can serve as guidance for future development of other teleconference groups.

METHODOLOGY

Vidyo® teleconference system is used. The running of the ECE teleconference is as described: twice technical test sessions are organized within 2 weeks prior to the event; total time of 90±10minutes is allotted for each meeting and 7-9 minutes for each presentation; concurrent communication is established among the engineering team for troubleshooting.

Country's economic situation, time zone difference from Japan, connectivity with research and education network and engineering cooperation of each member were individually described and analyzed with regards to their association with participation enthusiasm which was taken as participation of at least 50% of the meetings since their joining in. Association between these factors were calculated using two-way table with chi-square test to generate odds ratio(OR) and p-value.

RESULTS

Since the formation till May 2016, ECE members increased by 314%(from 7 to 29). Feedback received indicated high-level of satisfaction for program content, audio-visual transmission and ease of technical preparation. Time zone difference of more than 6 hours and poor engineering cooperation were independently associated with inactive participation [OR of 3.38 with 95% confidence interval(CI) of 1.89–6.04, p=0.04 and OR of 21.33 with 95% CI of 2.95-154.55, p=0.001 respectively].

CONCLUSIONS

Good program content and high quality audio-visual transmission are keys to the success of a medical teleconference. In our analysis, poor engineering cooperation and huge time zone difference contributed to inactive participation.

A PROSPECTIVE STUDY OF THE PREVALENCE AND CHARACTERIZATION OF ANTIBIOTIC RESISTANCE IN HELICOBACTER PYLORI STRAINS IN QUEEN ELIZABETH HOSPITAL (QEH), SABAH, MALAYSIA

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BACKGROUND & OBJECTIVES

There is a high prevalence rate of H.Pylori in Sabah amongst the indigenous populations. However local data on H. Pylori resistance pattern is lacking in this part of Borneo. The objective of this study is to determine the prevalence of antibiotic resistance in H.Pylori strains in Sabah. We also aimed to estimate the mean minimum inhibitory concentration (MIC) of clarithromycin, metronidazole, tetracycline, amoxicillin and ciprofloxacin.

METHODOLOGY

This study was conducted from December 2014 and is still ongoing. Samples were collected from subjects who tested positive for H.Pylori infection during endoscopy. Those samples with successful isolation of H. Pylori underwent antibiotics susceptibility testing using the E-test method.

RESULTS

As an ongoing study, we have successfully isolated H.Pylori strain from 52 patients thus far. The mean age was 52 years (SD15). 55% of patients were of Kadazan-Dusun ethnicity. The commonest clinical presentation was epigastric pain (52%). The commonest endoscopy finding was erythematous pangastritis (73%). Ten patients (19%) tested positive for antibiotics resistance. The prevalence of antibiotics resistance was 15.4% for metronidazole and 3.8% for clarithromycin and ciprofloxacin each. There was no resistance detected for amoxicillin and tetracycline. Two patients had multiple drug resistant (MDR) strains. The MDR strains were associated with higher MIC levels as compared to the single drug resistant group. The median MIC in patients with sensitive strains are: amoxicillin 0.016ug/ml(IQR 0.002), metronidazole 0.38ug/ml(IQR 0.719), clarithromycin 0.023ug/ml(IQR 0.026), tetracycline 0.125ug/ml(IQR 0.026) and ciprofloxacin 0.125 ug/ml(IQR 0.145). In the patients with antibiotic resistance, the median MIC for metronidazole was 152ug/ml(IQR 230); mean for clarithromycin 20ug/ml(SD 17) and ciprofloxacin 22ug/ml(SD 14).

CONCLUSION

Based on our preliminary data, the prevalence of antibiotics resistance is still low in Sabah. However the high median MIC in the resistant group is cause of concern.

ACUTE PHLEGMONOUS GASTRITIS - A RARE CAUSE OF ACUTE ABDOMEN

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INTRODUCTION

Acute phlegmonous gastritis(PG) is an uncommon, rapidly progressive fatal systemic infection with clinical symptoms restricted predominantly to the stomach. The common factors in all of the survivors were the early recognition and the prompt antibiotic treatment or surgery.

CASE

A 63 year old lady presented with sudden onset severe continuous epigastric pain associated with abdominal distension, vomiting and high grade fever. On presentation, she had stable vitals with temperature of 380C. She had epigastric tenderness with sluggish bowel sounds. Her blood parameters revealed normochromic normocytic anemia, leukocytosis with elevated C-reative protein levels. She also had acute kidney injury. CT Abdomen done revealed a thickened and edematous greater and lesser curvature of stomach measuring 2cm in thickness. These features were suggestive of PG. She was kept nil by mouth, started on broad spectrum antibiotics and was given aggressive fluid hydration. She was also started on total parenteral nutrition. She gradually improved after four days. Initial OGDS done showed thickened edematous stomach body. Endoscopic ultrasound(EUS) revealed thickened stomach wall with poor differentiation of the stomach layers. The antral mucosa measured 5cm in thickness. Fluid aspirated for culture grew Acinetobacter sp, sensitive to Meropenem. She began tolerating orally eight days later and was subsequently discharged well.

DISCUSSION

There are two types of phlegmonous gastritis described- diffuse type and less frequently localized type involving the antrum. The possible route of infection is hematogenous and direct contamination. Diagnosis can be made by OGDS, CT scan or EUS. On CT scan, markedly thickened gastric wall can be seen, and collection of air in emphysematous cases. Commonly found pathogens on culture of gastric aspirates are streptococci. Phlegmonous gastritis can be treated conservatively with antibiotics and intravenous fluid infusion, if the disease is diagnosed early. Otherwise, surgical management should be considered.

PROSPECTIVE STUDY ON ENDOSCOPIC ULTRASOUND GUIDED FINE NEEDLE ASPIRATION CYTOLOGY (EUS-FNAC) IN QUEEN ELIZABETH HOSPITAL (QEH), SABAH

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OBJECTIVES

The study aim was to determine the epidemiology, indications and diagnostic yields of EUS-FNAC in QEH.

METHODOLOGY

This prospective study was carried out from September 2015 to April 2016 on all EUS procedures done in the Gastroenterology Unit at QEH.

RESULTS

Data was collected from a total of 48 patients. The median age was 58 years (IQR 24). 56% were male and 43% were female. The indications for FNAC were: pancreatic malignancy (45%), intra-abdominal lymphadenopathy (16.7%), liver lesions (12.5%) and CBD mass (10.4%). A total of 91 passes were made of which 36 (40%) were from lymph nodes, 30 (33%) from pancreas and 16 (18%) were from liver lesions. The positive diagnostic yield was 79%. The yield was highest for the FNA of pancreatic lesions (93%) of which 71% were adenocarcinoma. A large proportion of the pancreatic FNA were from the head of pancreas. The yield for FNA from liver lesions was 75% of which 67% were metastatic lesions. Diagnostic yield for lymph node FNA was lower at 67%. Of those with positive yield in the lymph node group, 29% were metastatic carcinoma and 16.7% were lymphoma. The number of passes made during FNA was mostly one pass (40%) or two passes (46%). There was a preference to use 22G needle (70%) as compared to 19G (16.5%) and 25G (13%). Using Fisher's exact test, there was no statistically significant difference seen between 19G vs 22G needle (p=0.736), 22G vs 25G needles (p=0.44) and between 1 pass vs 3 passes (p=1). Using Pearson Chi-Square, there was also no statistically significant difference between 1 pass vs 2 passes (p=0.729).

CONCLUSION

There were no statistically significant differences seen in our sample yield between different needles and number of passes. However the sample size was small and a proper randomized study is planned for the future.

TUBERCULOSIS IN DISGUISE - A RARE PRESENTATION WITH OBSTRUCTIVE JAUNDICE

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INTRODUCTION

Hepatobiliary tuberculosis is an extremely rare form of TB. The manifestations of biliary TB can often mimic that of malignancies.

CASE

A 26 year old registered nurse presented with a three days history of jaundice. Physical examination revealed a deeply jaundiced lady with no other remarkable signs. Blood investigations revealed a normochromic normocytic anemia (Hb 10g/dL) with raised inflammatory markers; elevated bilirubin levels (113umol/L) with elevated alkaline phosphatase (552U/L); and reversal in albumin-globulin ratio. An ultrasound and CT performed showed an echogenic lesion within the common hepatic duct (CHD) and main intrahepatic ducts (IHD) measuring 2.7x2.3x1.6cm with distal IHD dilatation. MRCP showed multisegmental stenosis of the IHD's and a long segment stenosis involving the hilar confluence. ERCP confirmed Bismuth IV hilar stricture. Plastic biliary stents were deployed into the right and left system (10F stents). Brushings were negative for malignancy. She was empirically started on antituberculos treatement. The bile MTB C&S subsequently was positive for tuberculosis. She had a total of 18 months of anti-tuberculous treatment based on her response. Repeat ERCP was performed three months later with biliary stenting of both right and left systems. After six months anti-tuberculous treatment, the plastic stents were removed as liver function normalized. The bilirubin has remained normal since then with an ALP level ranging from 180 to 240 U/L. She has remained asymptomatic two years post treatment.

DISCUSSION

In suspected cases of hepatobiliary TB, evaluation begins with clinical findings followed by radiological imaging. This is then followed by confirmatory test by tissue acquisition. Tissue samples can be obtained during ERCP (bile culture, brushing, biopsies), fine needle aspiration of nodes/masses during endoscopic ultrasound, ultrasound guided biopsy of concomitant liver lesions and laparascopic biopsy. The treatment of hepatobiliary TB includes antituberculous treatment with biliary decompression of dominant strictures with graded dilatation.

SERUM CATHEPSIN D AND CYTOKERATIN - 18 LEVELS FOR LOBULAR INFLAMMATION AND BALLOONING IN ADULT NAFLD PATIENTS: NON INVASIVE BIOMARKERS IN DIAGNOSIS OF NON ALCOHOLIC STEATOHAPETITIS(NASH) IN ADULT PATIENTS

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INTRODUCTION

Lobular inflammation and ballooning are major indicators towards the diagnosis of NASH. Serum cathepsin-D (CatD) and cytokeratin-18 (CK-18) levelswere studied in adult patients with biopsy-proven NAFLD.

METHODS

Corresponding data on serum CatD(pg/mL) and CK-18 levels(U/L) for 217 liver biopsies of adult NAFLD patients were analyzed. The biopsies were graded according to the NASH Clinical Research Network Scoring System. Lobular inflammation was dichotomized to none or mild (grades 0-1) vs. more severe (grades 2-3), and ballooning was dichotomized to none (grade = 0) vs. few or many (grades 1-2).

RESULTS

CatDwas not significantly higher in subjects with more severe inflammation [315 (226-452) vs. 366 (249-521), p=0.296]. CatDwas significantly higher in subjects withballooning compared with those without ballooning [371 (255-522) vs. 289 (212-390), p=0.005]. The AUROC for CatDto diagnose ballooning was 0.62. Using 353 as the optimal cut-off,the sensitivity, specificity, positive predictive value and negative predictive value for CatD to diagnose ballooning was 53.8%, 74.6%, 85.0% and 37.6%, respectively.CK-18 was significantly higher in subjects withmore severe inflammation[416 (231-627) vs. 295 (194-479), p=0.01]. The AUROC for CK-18 to diagnose more severe inflammation was 0.60. Using 536 as the optimal cut-off,the sensitivity, specificity, positive predictive value and negative predictive value for CK-18 to diagnose more severe inflammation was35.1%, 84.9%, 65.4% and 61.6%, respectively.CK-18was significantly higher in subjects with ballooning compared with those without ballooning [377 (219-573 vs. 279 (178-434), p = 0.006]. The AUROC for CK-18to diagnose ballooning was 0.62. Using the 324 optimal cut-off,the sensitivity, specificity, positive predictive value and negative predictive value of CK-18 to diagnose ballooning was 57.1%, 66.7%, 81.7% and 37.4%, respectively.

CONCLUSION

CatD and CK-18 do not appear to be useful for diagnosis of lobular inflammation and ballooning in adult NAFLD patients.

TREATMENT OUTCOMES WITH PEGYLATED INTERFERON AND RIBAVIRIN IN TREATMENT OF CHRONIC HEPATITIS C PATIENTS: HOSPITAL PULAU PINANG EXPERIENCE

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OBJECTIVE

To evaluate the treatment outcomes with pegylated interferon and ribavirin in chronic Hepatitis C (CHC) patients in Hospital Pulau Pinang.

METHODOLOGY

This is a retrospective study of treated CHC patients. Demographic data and treatment outcomes were collected retrospectively from medical records.

RESULTS

A total of 136 patients (81 males, 56 females) were treated. There were 25 Malays (18.4%), 96 Chinese (70.1%), 12 Indians (8.8%) and 3 of other ethnicity (2.7%). 39 (28.7%) patients were genotype 1, 93 (68.4%) patients were genotype 3 and 4 (2.9%) patients were genotype 2. In genotype 1 hepatitis C virus (HCV) infection, 25 patients achieved SVR (64.1%) and 14 failed (35.9%). Majority of patients who achieved sustained virological response (SVR) (76%) had no liver cirrhosis and only 6 of them had liver cirrhosis (24%). Out of those who failed to achieve SVR, 8 were relapsed and 6 were non-responders. 2 (25%) relapsed patients and 3 (50%) non-responders had liver cirrhosis. In genotype 3 HCV infection, 66 patients (71%) achieved SVR and 27 patients (29%) failed. Majority of patients who achieved SVR (86.4%) had no liver cirrhosis. For those who failed to achieve SVR, 24 of them relapsed and 3 were non-responders. 17 (70.8%) of the relapsed and one (33.3%) of the non-responder had liver cirrhosis. All the 4 (100%) genotype 2 HCV infection patients achieved SVR and none of them had liver cirrhosis.

CONCLUSION

In our experience, 60% to 70% patients treated with pegylated interferon and ribavirin achieved SVR. The rate differs and depends on the viral genotype in which genotype 3 HCV patients had higher SVR rate. Patients who had no liver cirrhosis had higher SVR rate across all the genotypes. This implies that patient without liver cirrhosis responds better to treatment.

DYSPEPSIA IN NSAID USERS WITH OR WITHOUT PREVENTIVE MEASURES - A LONGITUDINAL STUDY

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BACKGROUND

NSAID-induced dyspepsia, the most common minor gastrointestinal side effect of NSAIDs, has not been widely studied as compared to NSAID-related gastropathy. This study aimed to characterise dyspepsia in regular NSAID users, and assess the effects of and adherence to preventive measures prescribed.

METHODS

A longitudinal study was conducted in the Outpatient Pharmacy of a large, teaching hospital in Malaysia. All patients who met the inclusion criteria for regular NSAID use were recruited. Participants were interviewed using a locally validated version of the Leeds Dyspepsia Questionnaire. Participants were followed-up via telephone interview for 2 weeks, whereby their dyspepsia symptoms were again assessed, and adherence to any prescribed preventive measures was evaluated using the 8-item Morisky Medication Adherence Scale. In this study, preventive measures for dyspepsia included histamine-2 receptor antagonists, proton pump inhibitors, antacids and cyclo-oxygenase (COX)-2 inhibitors.

RESULTS

Of the 409 participants recruited, 60.6% were naïve and 39.4% were repeated NSAID users. The mean (standard deviation) age of the participants was 52.3(14.6) years, with 60.6% female. The types of NSAIDs used were: 59.2% non-selective NSAIDs, 39.8% COX-2 inhibitors and 1% combination of NSAIDs. At baseline, 54.4% of naïve and 45.3% of repeated NSAID users had dyspepsia. Among the 190 naïve NSAID users, more participants without preventive measures experienced worsening of dyspepsia at Week 2 ($p=0.044$) while more participants with preventive measures experienced improvement of dyspepsia ($p=0.039$). Of the 53 participants who received preventive measures and were analysed for adherence, 49.1% were non-adherent at Week 2 and the most common reason cited was the absence of gastrointestinal symptoms (80.8%).

CONCLUSION

Dyspepsia is common in patients on regular NSAID use, regardless of the type. Its severity can be reduced considerably by the use of preventive measures. However, patient adherence to such preventive measures was suboptimal and needs to be emphasized.

INAPPROPRIATE PRESCRIPTIONS OF PROTON PUMP INHIBITORS FOR STRESS ULCERS PREVENTION AMONG MEDICAL INPATIENTS

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INTRODUCTION

Proton pump inhibitors(PPI) are commonly used for treatment and prophylaxis of various peptic ulcer related conditions,including stress ulcers. However, there are various reports about their inappropriate use for stress ulcer prevention.

OBJECTIVE

To evaluate the appropriateness of PPIs prescription by conducting an audit of medical inpatients against recommended guidelines.

METHODS

We carried out a retrospective,analytical study from January to February 2016 in the general medical wards of a local tertiary hospital. Records of drug prescriptions from the hospital pharmacy were used to identify all patients who received PPI during hospital stay. Samples were chosen randomly from these inpatients.The indication of PPI application was defined according to American Gastroenterology Association(AGA) guideline.

RESULT

A total of 200 patients were recruited randomly. PPIs were prescribed for 42% of patient without any proper indication. The estimated cost for inappropriate use of PPIs was 500 000 Ringgit Malaysia per year.

CONCLUSION

We reported a high frequency of inappropriate prescriptions of PPIs among our patients. A more rational use of PPIs will improve both health care cost and patient safety.

KEYWORDS

Inpatients,Inappropriate Prescriptions,Proton Pump Inhibitors,Peptic Ulcer

SHORT-TERM COMPLICATIONS ASSOCIATED WITH NASOGASTRIC VERSUS PERCUTANEOUS ENDOSCOPIC GASTROSTOMY FEEDING IN OLD PATIENTS WITH DYSPHAGIA – PRELIMINARY RESULTS FROM A PRAGMATIC STUDY

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BACKGROUND

Qualitative studies in Malaysia have suggested that clinicians managing older patients with dysphagia are not convinced of the benefits of percutaneous endoscopic gastrostomy (PEG) over long-term nasogastric (NG) tube feeding.

OBJECTIVE

To compare clinical outcomes in dysphagic older Asian subjects with either NG tube or PEG tube feeding using a pragmatic study design.

METHODOLOGY

Consecutive adults referred for PEG feeding were recruited together with controls on long-term NG feeding. Patients were assessed and followed up for three weeks, two months and four months after enrolment. Short-term complications are reported here.

RESULTS

102 participants (NG fed=52, PEG fed=50) were recruited over 2 years from 2013 - 2015. There were significant differences in age at baseline (82.67±7.15 vs 76.88±7.37; p = <0.001) but not gender or other parameters. At three weeks' follow-up 10 individuals in the NG arm and 2 individuals in the PEG arm had died. Follow-up information at three weeks was available for 41 individuals in the NG group and 43 individuals in the PEG group. After adjustment for confounders such as age, survivor analysis between NG and PEG group revealed significant tube-related complications (NG 42% versus PEG 14%, p=0.047) and gastro-intestinal complications (NG 12% versus PEG 2%, p=0.042) at three weeks in the former. However, no significant difference in overall complications was observed between both group of patients (p=0.533).

DISCUSSION

Preliminary results from this study indicate that PEG feeding is associated with fewer tube-related complications and no increase in overall complications compared to NG feeding even in the short-term. Our findings should allay the fears of many healthcare professionals over the short-term complications associated with PEG feeding.

NON-STEROIDAL ANTI-INFLAMMATORY DRUG (NSAID)-INDUCED GASTROINTESTINAL ADVERSE EFFECTS IN ADULTS WITH CHRONIC RHEUMATOLOGICAL DISORDERS - A MULTI-CENTRE, RETROSPECTIVE, COHORT STUDY

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BACKGROUND

Adults with rheumatological diseases on long-term non-steroidal anti-inflammatory drugs (NSAID) are at risk of GI adverse events, but data among Asian subjects are lacking. This study aims to describe the prevalence and predictive factors for GI adverse events from a large cohort of such patients in Malaysia.

METHODS

A retrospective cohort study was conducted between 2010 and 2014. Computerised databases of clinical records and pharmaceutical prescriptions from 4 of the main rheumatology units in this country were reviewed. Long-term NSAID therapy was defined as a minimum duration of 4 weeks.

RESULTS

Data on 634 rheumatological patients were included in the final analysis with the following characteristics: mean age 53.4 ± 12.5 years, 89.9% female, diagnosis: rheumatoid arthritis (RA) 59.5%, osteoarthritis (OA) 10.3% and RA/OA combination 30.3%. 286 (45.1%) patients were on regular Prednisolone therapy, with long-term NSAID therapy as follows: cyclo-oxygenase (COX)-2 inhibitors n=263 (41.5%) and non-selective NSAID n=371 (58.5%). The number of GI risk factors for the cohort were as follows: none n=241(38%), one n=302(47.6%), two n=79(12.5%) and three n=12(1.9%). There were a total of 84 (13.2%) GI adverse events during the period of study, with details as follows: abdominal pain/ gastritis 91.6%, gastro-duodenal ulceration 6.0% and bleeding gastric ulcer 2.4%. Multivariate analysis subsequently revealed that the following factors were independently predictive of a GI adverse event: a previous history of GI disease (OR 6.9, 95% CI=3.2-14.8), Prednisolone therapy (OR 5.6, 95%CI=2.8-11.3) and > 1 GI risk factor (OR 8.6, 95% CI=3.9-18.5). Interestingly, the type and number of NSAIDs did not influence GI adverse events in this cohort of patients.

CONCLUSION

GI adverse events are not uncommon in Malaysian rheumatological patients on long-term NSAID therapy. Targeting at risk cases with appropriate gastric anti-secretory therapy may help to reduce this complication.

CASE SERIES: A RARE CAUSE OF RECURRENT MELAENA - DUODENAL METASTASES OF LUNG ADENOCARCINOMA

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INTRODUCTION

Upper gastrointestinal bleeding (UGIB) as a result of gastrointestinal metastases from lung cancer is especially rare as an initial presentation. Specifically, duodenal metastasis as primary presentation is very uncommon with commonly distant metastases site found in the adrenals, bone, liver and brain. Among small bowel metastasis, the jejunum is the most frequent site (50.9%), followed by the ileum (33.3%), and the duodenum (15.8%).

CASE REPORT

First patient is a 64 year-old man, active smoker, presented with melaena, hemoptysis, weakness, and weight loss for past 2 months. Oesophagogastroduodenoscopy (OGDS) showed multiple polypoidal mass with Forrest IIc and III ulcerated centre at the second part of duodenum. Histopathological examination was typical for lung adenocarcinoma with immunostaining positive for CK7, TTF1 and negative for CK20, LCA. CT scan revealed right upper lobe bronchogenic carcinoma with liver, mesenteric and contralateral lung metastases. Patient subsequently succumbed to sepsis.

Second patient is a 73 year-old lady, non-smoker, presented with weakness, exertional dyspnea, weight loss and recurrent melaena. OGDS showed large circumferential mass measuring 2.5cm with ulcerated base at third part of duodenum.

Histopathological examination confirms duodenal metastasis with poorly differentiated adenocarcinoma. CT scan showed left upper lobe lung mass with lymphadenopathies and liver metastases. Treatment initiated with radiotherapy (30Gy) and chemotherapy utilizing cisplatin-paclitaxel.

DISCUSSION

UGIB due to duodenal metastasis of lung cancer is rare. Reported gastrointestinal metastasis from lung cancer ranges from 0.2 to 11.9% but with only 0.2-0.5% showing symptoms. Abdominal pains are often mistaken as side effects of concurrent radiotherapy and chemotherapy. Faecal occult blood testing is useful as an initial screen. Current treatment modalities include pancreatiduodenectomy, endoscopic resection and chemo-radiotherapy. Standard treatment has yet to be formulated although most would advocate surgery for palliative intent.

CONCLUSION

UGIB as initial presentation of distant metastasis from lung adenocarcinoma is extremely rare but need to be considered in cases of recurrent bleed in suspected or proven lung neoplasia.

NO CHANGE IN THE ANATOMIC LOCATION OF COLORECTAL CANCER IN A MULTIRACIAL ASIAN POPULATION IN MALAYSIA

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OBJECTIVE

In a previous publication we reported that Colorectal Cancer (CRC) in our patients were overwhelmingly left sided.1 We aim to determine if there was any anatomical shift in the location of CRC after a 10 year interval period.

METHODOLOGY

In this retrospective study, we analysed all patients who underwent colonoscopy in the endoscopy unit of University Malaya Medical Centre and were diagnosed to have CRC from January 2013 till March 2016.

RESULTS AND DISCUSSION

Over 39 months, 305 patients were diagnosed to have CRC. 227 (74.4%) had left sided tumour, 73 (23.9%) had right sided tumour whereas 5 (1.6%). The median (IQR) age was 66 (56.5, 74.0). The male to female ratio was 1.42. Majority were of Chinese ethnicity (58.7%), followed by Malays, Indians and other ethnic groups (25.6%, 14.1% and 1.6%). Sub analysis amongst patients with right sided and left sided CRC is shown below:

	Right sided (n=73)	**Left sided** (n=227)
Age: median (IQR)	68 (58.5,75.5) years	66 (56,73) years
Gender: ratio	1.21	1.47
male (%)	40 (22.86)	135 (77.14)
female (%)	33 (26.4)	92 (73.6)
Ethnicity (%)		
Malay	13 (16.88%)	64 (83.11%)
Chinese	43 (24.43%)	133 (75.57%)
Indian	17 (40.48%)	25 (59.52%)
Others	-	5 (100%)

CONCLUSIONS

There is a slight increase in the overall number of right sided CRC patients compared to the previous study1, however it isn't statistically significant. No difference was observed in age of presentation and gender distribution amongst both these subgroups.

REFERENCE

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IS SILYMARIN USEFUL FOR THE TREATMENT OF NON-ALCOHOLIC FATTY LIVER DISEASE?: A META ANALYSIS

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OBJECTIVE

To determine the usefulness of silymarin for treatment of Non Alcoholic Fatty Liver Disease (NAFLD).

METHODOLOGY

Search terms "silymarin", "human" and "fatty liver" were used in the Cochrane and PubMed databases. Identified records were screened and full-text articles which could potentially be included were retrieved and assessed. Human studies which used silymarin for the treatment of NAFLD and had a control arm were included. Review Manager 5.3 was used for data analysis.

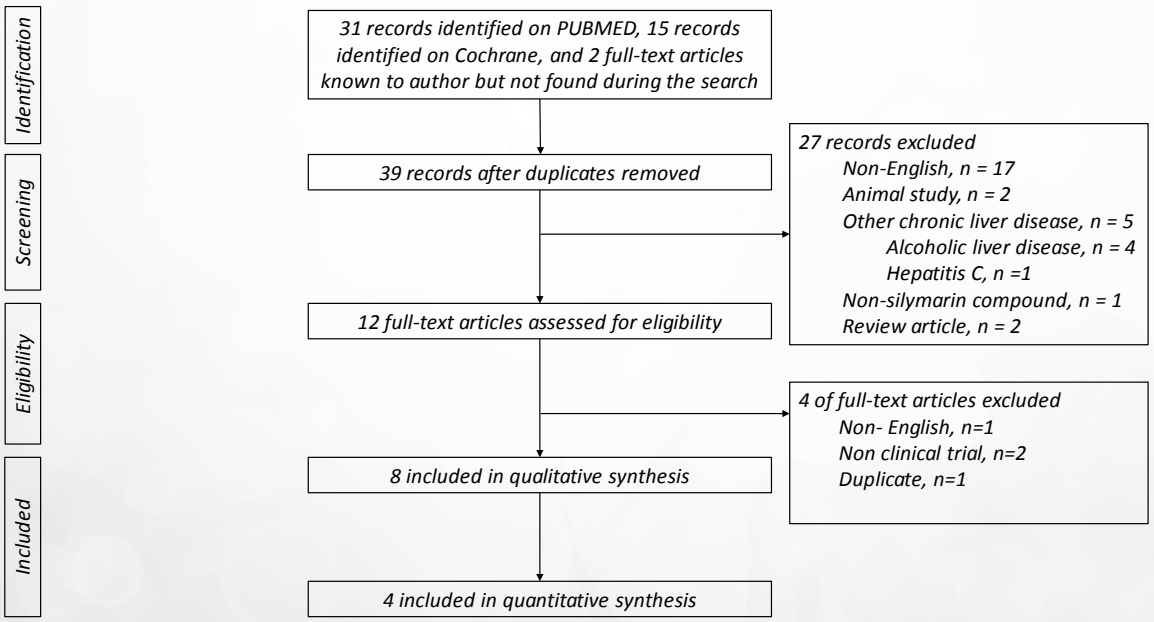
RESULTS

Out of 48 records identified, four were included in the meta-analysis (Figure 1). These studies were published between 2007 and 2015. A total of 301 patients were included, with 165 of them receiving silymarin between 2 to 6 months duration. The remaining 136 patients in the control arm were subjected to modified diet and physical activity and some were given placebo as well. The meta-analysis of 4 studies showed that the end-of-treatment serum alanine aminotransferase (ALT) level was significantly lower in the silymarin arm compared with the control arm[mean difference = -6.60, 95% CI= -12.15 – (-1.05), p=0.02]. The meta-analysis of 3 studies showed that the end-of-treatment serum aspartate aminotransferase (AST) level was significantly lower in the silymarin arm compared with the control arm [mean difference = -3.59, 95% CI= -7.12 – (-0.05), p = 0.05].

CONCLUSION

Using serum ALT and AST level as surrogate markers, silymarin appears to be useful for the treatment of NAFLD. Further studies on silymarin using histology or more accurate non-invasive methods for assessment of severity of NAFLD is warranted.

Figure 1 : Flow chart indicating selection process for this meta-analysis



EPIDEMIOLOGY OF LIVER CIRRHOSIS IN HOSPITAL TUANKU FAUZIAH, KANGAR

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OBJECTIVE

To identify the epidemiology of our liver cirrhosis patients in Hospital Tuanku Fauziah.

METHODS

This is a retrospective study involving patients who are or had attended our gastroenterology clinic over the last 7 years. Cards were traced and information extracted and analysed.

RESULTS

There were a total of 111 patients, of which 79 (70%) were male and 33 (30%) were female. Out of that 82 (75%) were Malay, 18 (16%) were Chinese, 3 (2.5%) were Indian, 6 (5.5)% were of Siamese descent and 1 of Bugis descent. The age of the patients ranged from 32 to 82 with the largest number of patients being in the 60 – 69 age group (n= 36, 32%). The most common cause of liver cirrhosis is hepatitis C (n= 40, 36%) followed by hepatitis B and cryptogenic cause (n=32, 29%) in which the majority of patients were diabetic while alcohol contributed to cirrhosis in 10% of the cases. 1 patient developed cirrhosis due to autoimmune hepatitis. At time of first diagnosis 62 (56%) patients were Child –Pugh classification A, 35 (32%) were Child's B and 13 (12%) were Child's C. Complications encountered by our patients were ascites in 40 (36%) people of which 7 (6%) developed spontaneous bacterial peritonitis. This was followed by esophageal varices in 33 (30%) . 13 (12%) of our patients were diagnosed to have hepatoma during the course of follow up with most diagnosed at age 50 – 70 years old. 5 patients had history of developing encephalopathy.

CONCLUSION

Viral hepatitis is still the commonest cause of liver cirrhosis but risk factors such as diabetes also poses a huge threat.

RISK OF LIVER INJURY ASSOCIATED TO BIOLOGIC DMARDS IN A SINGLE CENTRE STUDY FROM 2003 TO 2016

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Biologic DMARDs has been the mainstay for immunomodulation of autoimmune diseases like inflammatory bowel disease, rheumatic arthritis, psoriatic arthritis, spondyloarthropathy and others. There are well documented 4 types of liver injury associated to biologic DMARDs especially anti-tumor necrosis factor. This study was to review the effect of biologic DMARDs towards the liver function test in Selayang hospital over a period of 13 years. This is a retrospective study reviewing the liver function test of patients received biologic DMARDs which include anti tumour necrosis factor (namely infliximab, adalimumab, etanercept, Golimumab), monoclonal antibody anti CD 20 rituximab, interleukin 6 inhibitor tocilizumab, and Janus Kinase inhibitor tofacitinib. All data was retrieved from electronic system. There were a total of 99 patients followed up under rheumatology clinic of Selayang hospital with normal pre-screened prior to receive biologic DMARDs with or without methotrexate. The elevation of alanine aminotransferase (ALT) of more one times upper normal limit (ULN) as well as twice and more was compared. Elevation of alanine aminotransferase of more than 1x ULN is classified as elevated and more than twice is abnormal. Temporal occurrence of the liver derangement was studied. Of all the subjects, none of them develop acute liver failure. There was 1(1.0%) reactivation of hepatitis C; 31 subjects (31.0%) had transaminitis of more than 2 times ULN; 25 subjects (25.0%) more than one time ULN and 40 subjects (40.0%) were of normal. Of note the elevation of liver function test was transient. None of the subjects needed stoppage of biologic DMARDs. There are 5(5.0%) of patients needed their methotrexate stopped and liver derangement resolved though the biologic DMARDs were continued. In conclusion the usage of biologic DMARDs in our pool of patients was associated with transient raised of liver function and had not caused major serious liver disorder.

SYMPTOM IMPROVEMENT IN IRRITABLE BOWEL SYNDROME (IBS) WITH AN ASIAN LOW FODMAP DIET – A PILOT STUDY

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BACKGROUND

The efficacy of an Asian low fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs) diet in adults with irritable bowel syndrome (IBS) remains uncertain. We aimed to describe our early experience in a single-center with a dedicated Gastroenterology dietetic service.

METHODS

Consecutive patients with IBS referred to our dedicated Dietary Gastroenterology Clinic between February 2016 to May 2016 were screened. Data on demographic and clinical variables were obtained from patients' records and prospective telephone interviews. Information collected at the telephone interview included symptom improvement (abdominal pain, bloating, flatulence and stool formed pre and post-diet) following a minimum of 2 weeks compliance with a low FODMAP diet.

RESULTS

Thirty IBS patients had been commenced with a low FODMAP diet and 18 (60%) were available for a telephone interview.The respondents' characteristics were as follows: mean age 61 ± 16.9 years, 60% females, ethnicity 50% Chinese, 28% Indians and 22% Malays. IBS sub-types were as follows: IBS-D n=8, IBS-C n=1 and mixed n=9. Symptom improvement was reported in 5/9 (56%) cases with abdominal pain and in 15/15 (100%) cases with abdominal bloating. In terms of stool consistency, 9 patients had diarrhea and 8 reported more formed stool. Among 13 patients who had troublesome flatulence, 9 (69%) reported improvement and 4(31%) remained unchanged.

CONCLUSION

An Asian low FODMAP diet appears to be beneficial in patients with IBS, particularly among those with abdominal bloating and flatulence.

SERUM ASPARTATE AMINOTRANSFERASE LEVELS AS A PREDICTOR OF HISTOLOGICAL IMPROVEMENT IN PATIENTS WITH NON-ALCOHOLIC STEATOHEPATITIS: RESULTS FROM A RANDOMIZED, DOUBLE-BLINDED, PLACEBO-CONTROLLED STUDY

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BACKGROUND

In our randomized, double-blinded, placebo-controlled study on silymarin for the treatment of non-alcoholic steatohepatitis (NASH), histological improvement was seen in 51.2% (21/41) of subjects in the silymarin group compared with 28.6% (12/42) of subjects in the placebo group (p = 0.035). Histological improvement was defined as ≥ 2 points improvement in NAFLD activity score (NAS) without worsening fibrosis, or improvement in fibrosis regardless of change in NAS, in the end-of-treatment liver biopsy at Week 48 compared with the baseline liver biopsy.

OBJECTIVE

To study if serum aspartate aminotransferase (AST) level could predict histological improvement.

METHODOLOGY

The serum AST levels at Weeks 12, 24 and 48 compared with baseline for subjects with and without histological improvement were analyzed. Results: Serum AST decline at Week 12 was not associated with histological improvement (OR = 0.8, 95% CI = 0.3 – 2.1, p = 0.665). However, subjects with serum AST decline at Week 24 were more likely to have histological improvement compared with subjects without serum AST decline at Week 24 (OR = 21.3, 95% CI = 2.7 – 168.9, p = 0.004). The sensitivity, specificity, positive predictive value and negative predictive value for serum AST decline at Week 24 to predict histological improvement was 97.0%, 40.0%, 51.6% and 95.2%, respectively. Subjects with serum AST decline at Week 48 were more likely to have histological improvement compared with subjects without serum AST decline at Week 48 (OR = 7.3, 95% CI = 2.0 – 27.3, p = 0.004). The sensitivity, specificity, positive predictive value and negative predictive value for serum AST decline at Month 12 to predict histological improvement was 90.3%, 44.0%, 50.0% and 88.0%, respectively.

DISCUSSION AND CONCLUSION

Absent of decline in serum AST level at Weeks 24 and 48 appeared useful as a simple non-invasive test to identify NASH patients without histological improvement.

HELICOBACTER PYLORI PREVALANCE AND RESISTANCE PATTERN FOR PATIENTS IN HOSPITAL KUALA LUMPUR FROM JUNE 2014 TILL JUNE 2016

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OBJECTIVES

Toevaluate Helicobacter Pylori infection prevalence and resistance pattern among HKL patients in the last 2 years.

METHODOLOGY

A retrospective study which include the patients who had done OGDS with rapid urease tests(RUT) and urea breath tests(UBT). Results of patients underwent OGDS for tissue culture and sensitivity for Helicobacter Pylori were also reviewed.

RESULTS

A total of 5312 tests for H. Pylori were performed (864 UBT and 4448 RUT). 504 patients were positive for H. Pylori (9.5%). Majority of patients received amoxicillin, clarithromycin and proton pump inhibitors (PPI) twice daily dosing for 1 week unless penicillin allergic patient would received metronidazole. Out of these infected patients, 44 patients (8.7%) failed treatment ie tested positive with UBT at least 1 month after completed treatment. During the same period, 44 patients(those failed second line treatment including patients referred from other hospital) had OGDS guided biopsy done for culture and sensitivity for H. Pylori. Only 14 patients sample gave positive yield. From these results, resistance to both metronidazole and clarithromycin showed similar finding ie 57.1 % (8/14), levofloxacin 21% (3/14) and none showed resistance to tetracycline.

DISCUSSION

The resistance to clarithromycin has increased over years. Fairly good in vitro sensitivity towards levofloxacin indicate that levofloxacin is a good option as second line treatment. Tetracycline remain a viable treatment option for truly treatment failure patients even though dosing frequency may affect the compliance.

CONCLUSION

More effective combination therapy is required for the true treatment resistant patients. However, compliance and cost remained as a challenging task for clinician.

POLYP DETECTION RATE AND ADENOMA DETECTION RATE FOR GASTROENTEROLOGIST AND TRAINEES PERFORMING SCREENING COLONOSCOPY FOR PATIENTS IN HOSPITAL KUALA LUMPUR FROM JANUARY 2015 TILL DECEMBER 2015

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OBJECTIVES

Toevaluate polyp and adenoma detection rate for gastroenterologist and trainee performing screening colonoscopy for asymptomatic patients.

METHODOLOGY

A retrospective study which include the patients who had screening colonoscopy from 1 January till 31 December 2015 using database from Malaysia Gastro-Intestinal Registry (MGIR).Only those age more than 50 years with average risk for CRC with complete colonoscopic examination with good to excellent bowel preparation were recruited. Histopathology reports of all polyps were reviewed.

RESULTS

5 gastroenterology endoscopists (2 consultants and 3 trainees with years of experience ranging from 10 years to 6 months) performed 111 screening colonoscopies (5 cases excluded as poor bowel preparation) out of total 883 colonoscopies done in 2015 (13%). Consultant A performed 12 cases with polyp detection rate (PDR) of 58%% (7/12) and adenoma detection rate (ADR) of 42% (5/12). Consultant B attained PDR of 72%% (18/25) and ADR of 48 % (12/25). Trainee C achieved PDR of 43% (13/30) and ADR of 33% (10/30). Trainee D recorded PDR 45%(10/22) and ADR of 32% (7/22) respectively. Trainee E achieved PDR of 71% (12/17) and ADR of 35% (6/17). All adenoma were tubular adenoma with low grade dysplasia (LGD) except one with tubullovillous adenoma with LGD and 1 adenocarcinoma. Majority polyps are located at right colon, sessile (only 5 pedunculated) and diminutive (size ≤ 5mm with only 8 polyps are large > 10mm with largest 15mm)

DISCUSSION

With proper training and supervision, trainee achieved comparable PDR/ADR with trainers. Limitation of this review are small numbers, short audit time, no documented scope withdrawal time, exact number of polyps for each patients(with multiple polyps) and different colonoscope were used for each endoscopist.

CONCLUSION

Continuous PDR/ADR monitoring and training is important to meet the target ADR as per ASGE guideline to ensure quality CRC screening.

ENDOSCOPIC FINDINGS IN PATIENT WITH DYSPEPSIA: DIABETES MELLITUS (DM) VS NON DIABETES MELLITUS

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OBJECTIVE

To compare the endoscopic findings between DM and non DM in patients with dyspepsia.

METHODOLOGY

We have retrospectively review the patients who had undergone oesophagealgastroduodenoscopy (OGDS) for dyspepsia from 1st January 2010 to 31 December 2015 from Malaysia Gastrointestinal Endoscopy Registry, a total number of 6096 OGDS was done and the prevalence of endoscopy diagnosis were analysed.

RESULT

There were a total of 6098 ODGS done (DM=873 and non DM=5225).There is higher prevalence of oesophagitis, peptic ulcer disease, and gastroduodenal erosion. In DM group, there are higher use of antiplatlet and higher number of ischemic heart disease.

Comparison between normal population and Diabetic mellitus population

No.	Endoscopic Diagnosis	Non Diabetes Mellitus	Diabetes Mellitus
1	Oesophagitis	25.2%	28.5%
2	Benign Gastric Ulcer	5.1%	7.8%
3	Gastric Erosion	9.1%	12.4%
4	Benign Duodenal Ulcer	2.5%	4.0%
5	Duodenal Erosion	0.9%	1.7%
6	Gastritis	61.3%	54.8%
7	Duodenitis	17.1%	20.2%
8	Hiatus Hernia	21.8%	21.2%
9	Positive Urease Test	12.0%	11.9%
10	Normal OGDS	12.1%	7.9%
11	Malignant lesion	0.7%	0.2%
12	Antiplatlet use	4.8%	18.2%
13	Ischemic heart disease	5.7%	21.8%

CONCLUSION

Higher prevalence of oesophagitis, gastroduodenal erosion or ulcers in diabetes mellitus patients may attributed by high use of antiplatelet due to various condition, notably, ischemic heart disease.

NOTES

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Start Nexavar® Early

- in intermediate HCC patients unsuitable for TACE[2-3]
- in intermediate HCC patients who have failed or are refractory to TACE[2-5]
- in advanced HCC patients with portal vein thrombosis or macrovascular invasion[6]

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ABBREVIATED PRESCRIBING INFORMATION

Brand name of product: Nexavar® 200 mg film-coated tablets

Approved name of the active ingredient: Each tablet contains 200 mg of sorafenib (274 mg sorafenib tosylate)

Indications: Hepatocellular carcinoma (HCC): Nexavar is indicated for the treatment of patients with hepatocellular carcinoma. **Renal cell carcinoma (RCC):** Nexavar is indicated for the treatment of patients with advanced renal cell carcinoma who have failed prior interferon-alpha or interleukin-2 based therapy or are considered unsuitable for such therapy. **Differentiated thyroid carcinoma (DTC):** Nexavar is indicated for the treatment of patients with locally advanced or metastatic, progressive, differentiated thyroid carcinoma refractory to radioactive iodine **Dosage and method of administration:** The recommended daily dose of sorafenib is 400 mg (2 x 200 mg tablets) taken twice a day, either without food or together with a low fat or moderate fat meal. For oral use. To be swallowed with a glass of water. **Contraindications:** Sorafenib is contraindicated in patients with known severe hypersensitivity to sorafenib or any of the excipients. Sorafenib in combination with carboplatin and paclitaxel is contraindicated in patients with squamous cell lung cancer. **Special warnings and special precautions for use: Pregnancy:** Women should avoid becoming pregnant while on therapy. Breastfeeding should be discontinued during sorafenib therapy. **Dermatological Toxicities:** Hand-foot skin reaction (palmar-plantar erythrodysaesthesia) and rash represent the most common adverse drug reactions with sorafenib. **Hypertension:** An increased incidence of hypertension was observed in sorafenib-treated patients. **Hemorrhage:** An increase in the risk of bleeding may occur following sorafenib administration. **Warfarin:** Infrequent bleeding events or elevations in the International Normalized Ratio (INR) have been reported in some patients taking warfarin while on sorafenib therapy. **Increased Mortality Observed with NEXAVAR Administered in Combination with Carboplatin/Paclitaxel and Gemcitabine/Cisplatin in Squamous Cell Lung Cancer:** The use of NEXAVAR in combination with carboplatin/paclitaxel is contraindicated in patients with squamous cell lung cancer. NEXAVAR in combination with gemcitabine/cisplatin is not recommended in patients with squamous cell lung cancer. **Wound healing complications:** In patients undergoing major surgical procedures, temporary interruption of sorafenib therapy is recommended for precautionary reasons. **Cardiac Ischemia and/or Infarction:** Temporary or permanent discontinuation of sorafenib should be considered in patients who develop cardiac ischemia and/or infarction. **QT interval prolongation:** NEXAVAR has been shown to prolong the QT/QTc interval which may lead to an increased risk for ventricular arrhythmias. When using NEXAVAR in these patients, periodic monitoring with on-treatment electrocardiograms and electrolytes (magnesium, potassium, calcium) should be considered. **Gastrointestinal perforation:** Gastrointestinal perforation is an uncommon event and has been reported in less than 1% of patients taking sorafenib. **Hepatic impairment:** Since sorafenib is mainly eliminated via the hepatic route, exposure might be increased in patients with severe hepatic impairment. **Hypocalcaemia:** When using sorafenib in patients with DTC, close monitoring of blood calcium level is recommended. **TSH Suppression in DTC:** When using sorafenib in DTC patients, close monitoring of TSH level is recommended. **Drug-Drug Interactions:** *UGT1A pathway:* Caution is recommended when administering sorafenib together with compounds that are metabolized/eliminated predominantly by the UGT1A1 pathway. *Docetaxel:* Caution is recommended when sorafenib is co-administered with docetaxel. Neomycin: Co-administration of neomycin may cause a decrease in sorafenib bioavailability. *Paclitaxel/ Carboplatin:* No dose adjustment is needed when co administration of paclitaxel and carboplatin with 3-day break in sorafenib. Caution is recommended when sorafenib is administered without a break with paclitaxel and carboplatin. *Capecitabine:* Co- administration of capecitabine may cause increases in capecitabine and 5FU exposure modestly. *Doxorubicin/Irinotecan:* Caution is recommended when sorafenib is co-administered with doxorubicin/irinotecan. *Midazolam, dextromethorphan and omeprazole:* Concomitant administration with sorafenib do not alter the exposure of these agents. **Undesirable effects:** The most important serious adverse reactions were myocardial infarction/ischaemia, gastrointestinal perforation, drug induced, hepatits, haemorrhage, and hypertension/hypertensive crisis. The most common adverse reactions were diarrhoea, fatigue, alopecia, infection, hand-foot skin reaction, rash. For more details, please refer to full prescribing information.

For further prescribing information, please contact: Bayer Co. (M) Sdn Bhd, B-19-1 & B-19-2, The Ascent Paradigm, No. 1, Jalan SS 7/26A, Kelana Jaya, 47301 Petaling Jaya, Selangor, Malaysia. Subject to medical prescription.

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