The Hong Kong Society of Gastroenterology
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June
2022

The Council

President Message

Welcome to our June 2022 Newsletter!

It is my great honour to be elected as the new President of the Hong Kong Society of Gastroenterology and I am extremely humble to lead the Society which has just celebrated its 40th Anniversary. I would like to take this opportunity to thank all the previous Presidents and council members, particularly our immediate past President Dr. Jodis Lam, for their tremendous efforts in making the many achievements that the Society has accomplished over the past 4 decades. On behalf of the Society, I would also like to express my heartfelt gratitude to Drs. Chi-Ming Lam and Wai-Fan Luk for their valuable contributions to the Society. Both of them would retire from the council after serving for 16 years and 12 years, respectively. Our new council would include new office bearers with Professor Siew C. Ng as the Vice President, Professor Walter W.K. Seto as Honorary Secretary, Dr. Yee-Tak Hui as new co-opted member and Dr. Wai-Cheung Lao to continue to serve as Honorary Treasurer.

The 5th wave of COVID-19 has dealt a heavy blow to Hong Kong, affecting all walks of life in the territory. With the continuous social distancing measures, all large-scale events are put on hold including our Annual Scientific Meeting which has been postponed to 7 July 2022. Further updates would be provided in our websites and please be reassured that our Society, like the rest of Hong Kong, would rise to the challenge and thrive.

Finally, I would like to thank Professor Siew Ng for editing this Newsletter; Dr. Aneel Bhangu, Professor Vincent W.S. Wong, Dr. Shannon M. Chan and Dr. Gloria K.Y. Chan for providing the scientific updates in this Newsletter; and last but not least, all the sponsors who rendered their continuous support to the Society.

I look forward to your participation, either in person or virtually (if still needed), in our coming Annual Scientific Meeting on 7 July 2022 and the 24th Joint Annual Scientific Meeting on 18 September 2022.

Professor Wai-Keung Leung
President, The Hong Kong Society of Gastroenterology

Scientific Updates

COVIDSurg – generating globally relevant data

Dr. Aneel Bhangu
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United Kingdom

Background of the COVIDSurg Collaborative

The National Institute for Health Research (NIHR) Global Health Research Unit on Global Surgery (GSU), funded by the UK Government, was established in 2017 to connect frontline surgical and non-surgical researchers around the world and conduct research within a global network. The NIHR GSU has been conducting cohort studies on a global scale and has developed research hubs in seven low-to-middle-income countries to conduct large-scale randomized controlled trials (RCTs).

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(high branched chain amino acids)

The INTEGRAL NUTRITIONAL SUPPORT for Liver Disease Patients

Multiple Benefits for HCC Patients Undergoing TACE*,[1]

Lower post-chemoembolization morbidity
17.1% (BCAA) vs 37.2% (Control) (P=0.039)

Higher serum albumin levels
34 g/L (BCAA) vs 29 g/L (Control) at 9 months (P=0.042)

Better quality of life, denoted by FACT-G scores
89 (BCAA) vs 84 (Control) at 12 months (P<0.05)

Lower serum bilirubin levels
20 μmol/L (BCAA) vs 30μmol/L (Control) at 6 months (P=0.026)

BCAA: branched chain amino acids; FACT-G: Functional Assessment of Cancer Therapy-General; HCC: hepatocellular carcinoma; TACE: transarterial chemoembolization

***Study design[1]: This was a randomized controlled trial in which patients undergoing chemoembolization for HCC were randomized to receive oral BCAA for up to four courses of chemoembolization (n=41) or did not receive any nutritional supplement (n=43). Morbidity, liver function, nutritional status, quality of life and long-term survival were compared between the two groups. The morbidity rate was the overall frequency of morbidity after only TACE session during the four TACE sessions.**

References: 1. Poon RT, et al. Aliment Pharmacol Ther. 2004;19:779-88.

Abbreviated Prescribing Information

Aminoleban® EN powder (ORAL NUTRIENTS) 50 g/package. **INDICATION:** Improvement of the nutritional state of chronic hepatic insufficiency patients including those with hepatic encephalopathy. **DOSAGE:** For adults, reconstitute one package in about 180 mL of water or warm water (approx. 200 kcal/200 mL) and ingest with meals three times a day. Dosage may be adjusted according to the age and symptoms. **CONTRAINDICATION:** History of hypersensitivity to any ingredient of this product. Allergy to milk. **WARNINGS AND PRECAUTIONS:** Not to be administered into a blood vessel. Establish dosage based on individual patient's current treatment status including dietary therapy. For pregnant women during the first 3 months of pregnancy, or women who intend to become pregnant, adjust dosage as necessary to achieve a reduction to less than 5,000 IU/day of vitamin A. For patients requiring restriction of water intake, concentration of reconstituted product may be increased to approx. 2 kcal/mL (reconstitute one package in approx. 80 mL of water). **ADVERSE REACTIONS:** Diarrhea, abdominal distention, nausea, vomiting, anorexia, epigastric pain, abdominal pain, hyperammonemia, increased blood glucose, hypo-potassemia, edema, ascites, headache, dull headache, skin rash, pruritus, heartburn, cheilitis, glossitis, feeling abnormal, feeling hungry, jaundice, signs of abnormal hepatic function, increased weight, thirst, vertigo, somnolence, anemia, decreased urine output, hot flush. **Please see the full Prescribing information for details which is available upon request. (HKOP_AMINOLEBAN EN API_HK revised Apr 2020)**

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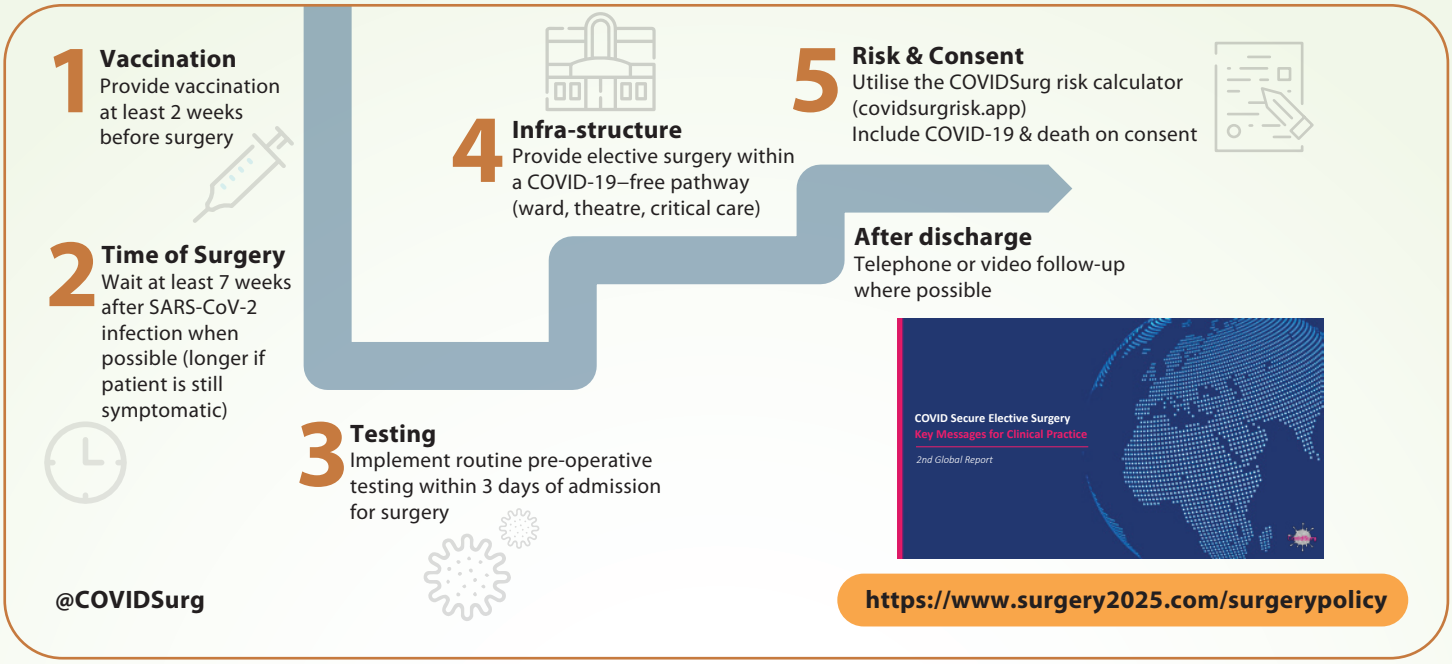
Figure. COVIDSurg: Summary of evidence-based, COVID-secure surgery pathway



In 2020, the NIHR GSU initiated COVIDSurg – a platform of studies that aimed to explore the impact of COVID-19 in surgical patients and services. Data generated from the COVIDSurg studies are of high quality and can provide data-driven guidance to surgeons around the globe during the pandemic.[1]

COVIDSurg helps implement safe surgery around the world

The first COVIDSurg study, published in The Lancet, was a cohort study in COVID-19–positive patients (N=1,128) who had surgery between January 1 and March 31, 2020, in 235 hospitals in 24 countries.[2] The study found that postoperative pulmonary complications occurred in 51% of patients with perioperative COVID-19 and were associated with a high 30-day mortality rate (38%). The pulmonary complication and mortality rates were higher than those reported for even the highest-risk patients before the pandemic.[2] The data suggested that surgery should be postponed, and non-operative management should be used whenever possible, during the pandemic.

Based on the COVIDSurg data, a pathway for patients from vaccination to the time of surgery was generated **(Figure)**. Key messages include:

1. Vaccination before surgery is necessary.
2. Patients should be tested for COVID-19 (PCR test) 3 days before a major surgery. If the result is positive, the surgery should be postponed for 7 weeks.
3. Preoperative isolation should be carefully considered before an elective surgery because it was associated with a higher postoperative pulmonary complication rate.
4. High-risk patients should be risk scored using the COVIDSurg risk calculator.
5. Any crucial surgery should be performed in a COVID-19–free pathway.
6. A high community COVID rate should be included in the consent form.

The first published COVIDSurg study received massive media coverage that crossed over into the public domain. Patient-facing materials were subsequently developed and are available in 22 languages to be utilized by healthcare professionals for patient education. Furthermore, accredited continuing professional development/continuing medical education (CPD/CME) platforms were launched, including www.surgery2025.com, where the lessons learned were harnessed into learning modules that are freely accessible to surgeons worldwide.[3]

In addition to cohort studies, COVIDSurg has also conducted other major RCTs, including the published RECOVERY trial, the soon-to-be-published FALCON trial and the ongoing CHEETAH, PENGUIN and GIRAFFE trials, all with fast recruitment and large sample sizes (>5,000 each). Moving forward, COVIDSurg will continue to advocate targeted data collection to support innovation in research and push research into practice through data publication and professional education platforms.

Conclusion

It is important for all surgeons to learn to identify excellent research from published papers to avoid implementing biased evidence into practice. Building on the global infrastructure provided by NIHR GSU, COVIDSurg delivers fast, global, high-quality research results and shortens the lag between data publication and practice change, allowing surgeons to bring the best benefit to patients.

References

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3. Surgery2025. Available at: https://modules.surgery2025.com/. Accessed 3 November 2021.

Current and future pharmacological treatment for NASH

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The Chinese University of Hong Kong
Hong Kong

NASH is a progressive form of NAFLD

Non-alcoholic fatty liver disease (NAFLD) is a major cause of chronic liver disease with a high prevalence of about 30% worldwide, including Asia.[1] Non-alcoholic steatohepatitis (NASH) – the active form of NAFLD – is characterized by hepatocyte injury, liver inflammation and progression of fibrosis.[2] It is an important cause of liver morbidity as the disease may progress to cirrhosis and hepatocellular carcinoma (HCC). Recent data from the US have revealed that NASH was the second most common indication for liver transplantation in 2019 and the fastest increasing indication. It was also the leading indication for liver transplantation among women without HCC.[3]

Weight reduction results in resolution of NASH

A 52-week prospective study of NASH patients (n=293) with a mean body mass index of 31.3 kg/m² showed that weight loss through lifestyle intervention caused resolution of NASH, and the rate of NASH resolution increased with increased percentage of weight loss. In patients who lost ≥10% of body weight, 90% had resolution of NASH and 81% had a reduction in fibrosis score.[4]

Weight reduction also predicts remission of NAFLD in non-obese patients. A community-based randomized controlled trial (n=154) demonstrated that half of non-obese patients achieved NAFLD remission with 3–5% weight reduction after 1 year of lifestyle intervention, but the same outcome required a 7–10% weight reduction in obese patients. Thus, modest weight reduction may be sufficient in non-obese patients.[5]

As weight reduction is difficult to sustain, especially in obese patients, many patients will need pharmacological treatment to control NAFLD, noted Professor Wong.

Potential pharmacological treatments for NASH

While there is currently no approved therapy, several drugs available for other indications have been studied for the treatment of NASH **(Table)**.[6]

Glucagon-like peptide-1 receptor agonists

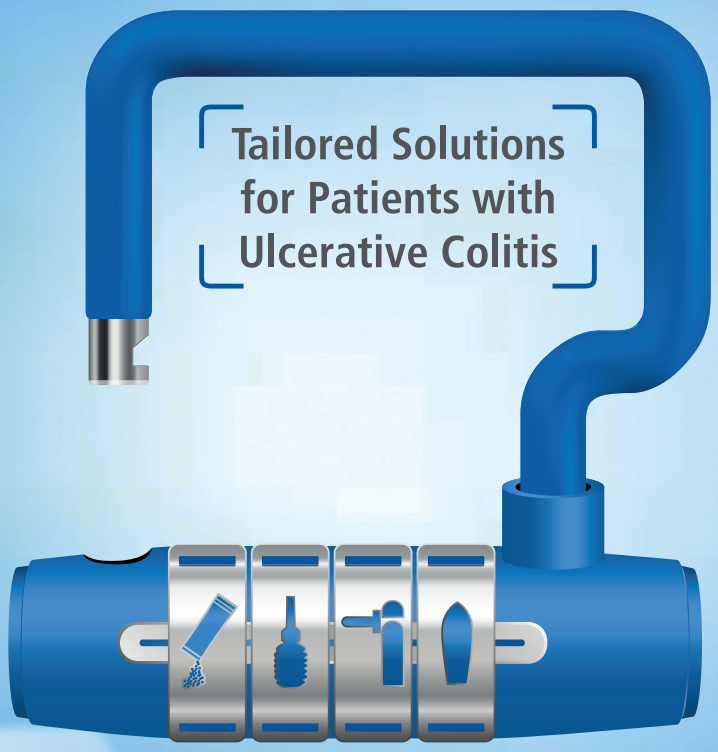
Several antidiabetic medications with cardioprotective and renoprotective benefits, eg, glucagon-like peptide-1 (GLP-1) agonists and sodium-glucose transport protein 2 (SGLT2) inhibitors, are being investigated in phase III trials for the treatment of NASH.[6]

Table. Potential use of off-label therapy* for NASH[6]

Agent	Effects on the liver	Other benefits	Key adverse events
Vitamin E	Improves hepatic steatosis and necroinflammation. May prevent liver decompensation and mortality.	Neutral metabolic effects.	May increase overall mortality at a high dose. May increase prostate cancer, heart failure and haemorrhagic stroke.
Pioglitazone	Improves hepatic steatosis and necroinflammation.	Improves insulin sensitivity and diabetic control.	Weight gain, fluid retention, bone loss, may increase bladder cancer.
GLP-1 agonist	Improves hepatic steatosis and necroinflammation.	Improves diabetic control, reduces major adverse cardiovascular events, weight reduction.	Nausea, vomiting, dyspepsia, diarrhoea and constipation.
SGLT2 inhibitor	Improves hepatic steatosis and necroinflammation.	Improves diabetic control, modest weight reduction. Prevents renal and cardiovascular events.	Genitourinary infection, acute kidney injury and euglycaemic diabetic ketoacidosis. May increase the risk of fractures and limb amputations.

GLP-1, glucagon-like peptide-1; NASH, non-alcoholic steatohepatitis; SGLT2, sodium-glucose transport protein 2
*These therapies are currently not approved by any regulatory authorities for the treatment of NASH. The information presented is solely for educational purpose. Before prescribing any products mentioned, please consult the Hong Kong-approved prescribing information issued by the manufacturer.

Tailored Solutions for Patients with Ulcerative Colitis



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Salofalk® mesalazine *4g/60ml Enema*

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Salofalk® mesalazine *Suppositories*

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*once daily dosing

References: 1. Kruis W, Kiudelis G, Rácz I, et al. Once daily versus three times daily mesalazine granules in active ulcerative colitis: a double-blind, double-dummy, randomised, non-inferiority trial. Gut. 2009;58(2):233-240. **2.** Bar-Meir S, Fidder HH, Faszczyk M, et al. Budesonide foam vs. hydrocortisone acetate foam in the treatment of active ulcerative proctosigmoiditis. Dis Colon Rectum 2003;46:929-936. **3.** Gross V, Bar-Meir S, Lavy A, et al. Budesonide foam versus budesonide enema in active ulcerative proctitis and proctosigmoiditis. Aliment Pharmacol Ther 2006;23:303-312.

Abridged Prescribing Information

Salofalk®

Salofalk® Granules 1000mg/3000mg; Salofalk® 250mg/500mg Gastro-resistant tablets, Salofalk® 250mg/500mg Suppositories, Salofalk® 4g/60ml Enema: Active ingredient: mesalazine (5-aminosalicylic acid). **Composition:** Active ingredient: 1 sachet of Salofalk® granules 1000mg/3000mg contains: 1000mg/3000mg mesalazine. 1 tablet of Salofalk® 250mg/500mg contains: 250 mg/500 mg mesalazine. 1 Salofalk® 250mg/500mg suppository contains: 250 mg/500 mg mesalazine. 1 enema of Salofalk® 4g/60ml contains: 4 g mesalazine. Other ingredients: See patient information leaflet. Note: Salofalk® enemas contain sodium benzoate and potassium metabisulphite. **Indications:** Salofalk® granules 1000mg/3000mg: Acute treatment and prevention of further episodes (for patients at increased risk for recurrence) of ulcerative colitis. Salofalk® 250mg/500mg tablets: Acute treatment and prevention of recurrence of ulcerative colitis. Acute treatment of Crohn's disease. Salofalk® 250mg/500mg suppositories: Acute treatment of ulcerative colitis confined to the rectum. Additionally, Salofalk® 250mg suppositories: prevention of recurrence of ulcerative colitis. Salofalk® 4g/60ml enemas: Acute treatment of ulcerative colitis. **Dosage:** Salofalk® granules 1000mg/3000mg: Adults: Acute episodes of ulcerative colitis: Up to 3000mg of mesalazine daily. Prevention of further episodes (for patients at increased risk for recurrence): 3000mg of mesalazine daily. Salofalk® 250mg/500mg tablets: Adults: recommended dose: Acute episodes of Crohn's disease: 500-1500mg mesalazine 3 times daily; Acute episodes of ulcerative colitis: 500-1000mg mesalazine 3 times daily; Prevent recurrence of ulcerative colitis: 500mg mesalazine 3 times daily. Children (≥ 6 years): starting dosage to be determined individually: Acute episodes: 30-50mg mesalazine/kg body weight/day in divided doses; Prevent relapse of ulcerative colitis: 15-30mg mesalazine/kg body weight/day in divided doses. Salofalk® 250mg/500mg suppositories: Adults: recommended dose: Acute episodes of ulcerative colitis: 500mg mesalazine 3 times daily; Prevent recurrence of ulcerative colitis: 250mg mesalazine 3 times daily. Salofalk® 4g/60ml enemas: Adults: recommended dose: 1 Salofalk 4g/60ml enema once daily. For further details on dosage: See patient information leaflet. **Contraindications:** Known hypersensitivity to salicylic acid, to salicylates or any of the excipients. Severe impairment of hepatic or renal function. **Pregnancy and lactation:** risk-benefit ratio. Additionally for Salofalk® Enemas: not to be used in case of sensitive patients (especially for known asthmatics or allergic anamnesis) due to the content of metabisulphite or sodium benzoate. **Side effects:** Headaches, dizziness, abdominal pain, diarrhea, flatulence, nausea, vomiting, chest pain, breathlessness or swollen limbs. Hypersensitivity reactions such as allergic exanthema, drug fever, dyspnoea. **Precautions:** History of lung problems. History of allergy to sulphasalazine. Hepatic or renal impairment. Concomitant use of azathioprine, 6-mercaptopurine or thioguanine, certain agents that inhibit blood clotting. Salofalk® 500mg suppository contains cetyl alcohol, which can cause local skin irritation. **Interactions:** See patient information leaflet. Please refer to package leaflet for further information. **Date of information:** 07/2017

Budenofalk® Rectal Foam

Active ingredient: budesonide. **Composition:** Each actuation of Budenofalk® rectal foam contains: active ingredient: 2 mg budesonide. Other ingredients: cetyl alcohol (Ph.Eur.), cetostearyl alcohol (Ph.Eur.), polysorbate 60, purified water, sodium edetate (Ph.Eur.), macrogolstearylether (Ph.Eur.) (10), propylene glycol, citric acid monohydrate. Propellants: butane, 2-methylpropane, propane. **Indications:** For the treatment of active ulcerative colitis limited to the rectum and the sigmoid colon. **Contraindications:** Hypersensitivity to budesonide or any of the other ingredients. Hepatic cirrhosis. Pregnancy. Lactation. Children. Close medical supervision is required in the following diseases: septicaemia, tuberculosis, hypertension, diabetes mellitus, osteoporosis, peptic ulcer (gastric or duodenal), glaucoma, cataract, family history of diabetes or glaucoma. Chickenpox, herpes zoster or measles. Local infections of the intestine (bacteria, fungi, amoebae, viruses). Severe hepatic impairment. Late stage primary biliary cirrhosis (PBC). **Side effects:** Urinary tract infections, anaemia, increase in erythrocyte sedimentation rate, leukocytosis, increased appetite, insomnia, dizziness, disturbances of smell, hypertension, nausea, abdominal pain, dyspepsia, flatulence, paraesthesias in the abdominal region, anal fissure, aphthous stomatitis, frequent urge to defecate, haemorrhoids, rectal bleeding, increase in transaminases (GOT, GPT), increase in parameters of cholestasis (GGT, AP), acne, increased sweating, increase in amylase, change in cortisol, burning in the rectum and pain, asthenia, increase in body weight. Side effects typical of systemic glucocorticosteroids may occur (the risk of side effects from Budenofalk® is generally lower). **Dosage:** Use 1 spray actuation daily, corresponding to 2 mg budesonide. Available on prescription only. **Date of information:** 02/2020

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HK/BUD/052022/209 (Jun 2022)

GLP-1 agonists mimic the effect of incretin (a gut-derived hormone), which stimulates insulin secretion, suppresses glucagon secretion, slows gastric emptying and reduces appetite by increasing satiety. GLP-1 agonists also reduce body weight and improve cardiovascular profiles.[2] In a phase II, double-blind study (n=320), semaglutide (a GLP-1 agonist) demonstrated the highest response rate in terms of NASH resolution ever reported for any pharmacological agent in patients with NASH and liver fibrosis. NASH resolution with no worsening of fibrosis was achieved in 40%, 36% and 59% of patients after 72 weeks of treatment with semaglutide 0.1 mg, 0.2 mg and 0.4 mg, respectively, compared with 17% in the placebo group ($p<0.001$ for semaglutide 0.4 mg vs placebo).[7] The study also revealed an inverse correlation between increasing semaglutide dose and the likelihood of fibrosis progression.[7]

Farnesoid X receptor agonist

Farnesoid X receptor (FXR), a nuclear receptor highly expressed in the liver, is involved in glucose and lipid metabolism. Preclinical studies have shown that obeticholic acid, a first-generation, potent FXR agonist, improved insulin sensitivity and had anti-inflammatory and antifibrotic effects in the liver. The 18-month interim analysis of a phase III randomized controlled trial (n=931) showed significant improvement in fibrosis (by ≥1 stage) in 23% and 18% of NASH patients with liver fibrosis who received obeticholic acid 25 mg and 10 mg, respectively, compared with 12% of patients who received placebo ($p=0.0002$).[8] Despite these findings, wider use of obeticholic acid may be limited by its side effects of pruritus and atherogenic dyslipidaemia, particularly at the daily dose of 25 mg.

Other agents

PF-05221304, an acetyl-CoA carboxylase inhibitor that targets de novo lipogenesis in the liver, has shown profound liver fat reduction in patients with NAFLD, compared with placebo, in a phase IIa randomized clinical trial.[9]

Lanifibranor, a pan–peroxisome proliferator-activated receptor agonist, has demonstrated efficacy in reducing disease activity without worsening of fibrosis in patients with active NASH in a phase IIb, randomized trial.[10]

Liver-directed, selective thyroid hormone receptor-β agonists, such as resmetirom, that increase hepatic fat metabolism with limited lipotoxicity are also being investigated for the treatment of NASH. Resmetirom resulted in hepatic fat reduction in patients with NASH in a 36-week phase II clinical trial.[11]

All these drugs are undergoing further assessment in phase III trials for NAFLD.

Of note, phase II studies of "pure" anti-inflammatory drugs, such as simtuzumab, selonsertib and cenicriviroc, have not shown improvements in hepatic inflammation or fibrosis in patients with NASH. These drugs may not be effective as monotherapy because the root causes of NASH, ie, obesity or metabolic dysfunction, are not being addressed, commented Professor Wong.

Conclusion

Weight reduction is associated with NASH resolution, but most patients will also need pharmacological treatment. A good metabolic drug with beneficial effects on body weight and metabolism would be preferred not only for treating NASH but also the related cardiovascular and metabolic outcomes. Upstream metabolic drugs should be the backbone of NASH treatment.

References

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Scientific Updates

The use of artificial intelligence in gastrointestinal endoscopy

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What is artificial intelligence?

Artificial intelligence (AI) refers to the ability of a computer to perform a task associated with intelligent beings, including 'cognitive' functions that might mimic the human mind, such as the ability to 'learn'. AI has evolved over the past few decades and, with 'deep learning' based on deep (artificial) neural networks inspired by the mammalian brain, has dramatically improved the efficiencies of workplaces and amplified what humans can do. It has also had a great impact in the field of medicine.

Application of AI in gastroenterology

Teaching or learning advanced endoscopy, such as magnifying endoscopy, takes a long time and very few people have excellent skills in this technology. The advent of AI has helped transfer the expertise of a few to the hands and minds of many.

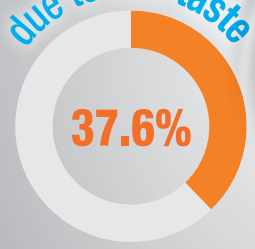
PICOPREP®

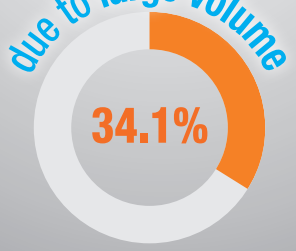
sodium picosulfate + magnesium oxide + citric acid

AIM FOR HIGH QUALITY BOWEL CLEANSING[1]

Bowel preparation has been considered the worst part of colonoscopy[2]

Reasons for patients not consuming the entire preparation:[3]





2 convenient dosing regimens to fit different patient needs:[4]

Morning Colonoscopy "Day-Before" Dosing

Afternoon Colonoscopy "Split-Dose" Dosing

300 mL (150 mL x2)[4]
Low-Volume Preparation

Easy-to-Take

Orange-Flavoured[4]

Improves Tolerability[5]

References:
1. Kojecky V, Matous J, Keil R, *et al. Dig Liver Dis.* 2018;50:271–276. 2. Dykes C, Cash BD. *Gastroenterol Nurs.* 2008;31:30–35. 3. Waldmann E, Penz D, Majcher B, *et al. United European Gastroenterol J.* 2019;7:114–124. 4. Hong Kong Product Package Insert of PICOPREP (Date of revision: AUG 2017). 5. Kojecky V, Matous J, Keil R, *et al. Int J Colorectal Dis.* 2017;32:1763–1766.

Abbreviated Prescribing Information of PICOPREP

Active Ingredient: Sodium picosulfate, light magnesium oxide, anhydrous citric acid. **Indications:** To clean the bowel prior to X-ray exam/ endoscopy, or surgery when judged clinically necessary. **Dosage and Administration:** Adult *Split-dose regimen (Preferred method)* 1st dose: 1 reconstituted sachet at the evening (e.g. 5-9 PM) of the day before procedure, followed by at least five 250 mL drinks of clear liqd w/in 5 hr before bed. 2nd dose: 1 reconstituted sachet 5 hr before procedure, followed by at least three 250 mL drinks of clear liqd w/in 5 hr up until 2 hr before procedure. *Day-before regimen (Alternative method)* 1st dose: 1 reconstituted sachet in the afternoon or early evening (e.g. 4-6 PM) before procedure, followed by at least five 250 mL drinks of clear liqd w/in 5 hr & before the next dose. 2nd dose: 1 reconstituted sachet 6 hr after 1st dose in the late evening (e.g. 10 PM-12 AM) followed by three 250 mL drinks of clear liqd w/in 5 hr before bed. Child 1st dose: Before 8 AM the day before procedure (≥ 9yr 1 sachet, 4-9 yr 1 sachet, 2-4 yr ½ sachet, 1-2 yr ¼ sachet). 2nd dose: 6-8 hr after 1st dose (≥ 9yr 1 sachet, 4-9 yr ½ sachet, 2-4 yr ½ sachet, 1-2 yr ¼ sachet). Clear liqd should include a variety of fruit juice without pulp, soft drinks, clear soup, tea, coffee (without milk, soy or cream) and water. Do not drink only water. **Contraindications:** Hypersensitivity, CHF, gastric retention, GI ulceration, toxic colitis, toxic megacolon, ileus, nausea & vomiting, acute surgical abdominal conditions, known or suspected GI obstruction or perforation, severe dehydration, rhabdomyolysis, hypermagnesemia, active IBD, severe renal impairment. **Special Warnings and Precautions:** Reconstitute right before administration; do not prepare the soln in advance. Prompt corrective action should be taken to restore fluid/electrolyte balance in patients with signs or symptoms of hypokalaemia or hyponatremia. Care should be taken in patients with recent GI surgery, heart diseases, IBD, reduced kidney function, on a controlled potassium diet, or on a regularly prescribed oral medication. Period of bowel cleansing should not be >24 hr. Early morning procedure may require 2nd dose during the night which may lead to sleep disturbance. Contains lactose, should not be used in patients with galactose intolerance, Lapp lactase deficiency or glucose-galactose malabsorption. Should not be used as a routine laxative. Avoid in pregnancy. **Side Effects:** Headache, nausea & proctalgia. **Interactions:** May modify absorption of other orally administered medicines (e.g. antiepileptics, contraceptives, antidiabetics & antibiotics). Chelation of Mg in PICOPREP w/ tetracycline & fluoroquinolone, Fe, digoxin, chlorpromazine & penicillamine; administer ≥ 2 hr before or ≥ 6 hr after taking PICOPREP. Reduced efficacy w/ bulk-forming laxatives. Increased risk of hypokalaemia w/ diuretics, corticosteroids or cardiac glycosides. Water retention &/or electrolyte imbalance w/ NSAIDs, drugs known to induce SIADH e.g. TCAs, SSRIs, antipsychotic drugs & carbamazepine.

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GA032/21/JUN/HK Jun/2021

In gastrointestinal (GI) endoscopy, AI can be used in detection and characterization of cancers and polyps and quality control.

Cancer detection with oesophago-gastro-duodenoscopy and AI
Two recently published studies demonstrated the ability of AI-based diagnostic systems with convolutional neural networks (CNNs) to detect pharyngeal cancer from still and video endoscopic images (white light or narrow-band imaging) with high sensitivity.[1,2]

Studies that compared the diagnostic results for oesophageal squamous cell carcinoma (ESCC) by deep learning-based AI systems and endoscopists (senior, mid and junior level) revealed that AI outperformed endoscopists regardless of experience level in terms of sensitivity, specificity, accuracy, positive predictive value and negative predictive value and improved diagnostic performance in screening of early ESCC.[3,4]

Using endoscopic images from patients with superficial ESCC, a study showed favourable performance of a deep learning-based AI system in diagnosing invasion depth and differentiating pathological mucosal and submucosal microinvasive (SM1) cancers from submucosal deep invasive (SM2/3) cancers with high sensitivity, specificity, positive predictive value, negative predictive value and accuracy that were comparable to experienced endoscopists.[5]

Furthermore, early oesophageal neoplasia in Barrett's oesophagus can also be detected by a CNN system with high sensitivity, specificity and accuracy. The object detection algorithm was also able to draw a localization box around the areas of dysplasia with high precision and at a speed that allowed real-time implementation.[6] Nevertheless, quality images are necessary for the precision, which still relies heavily on good control of the machine by trained human hands, commented Dr. Chan.

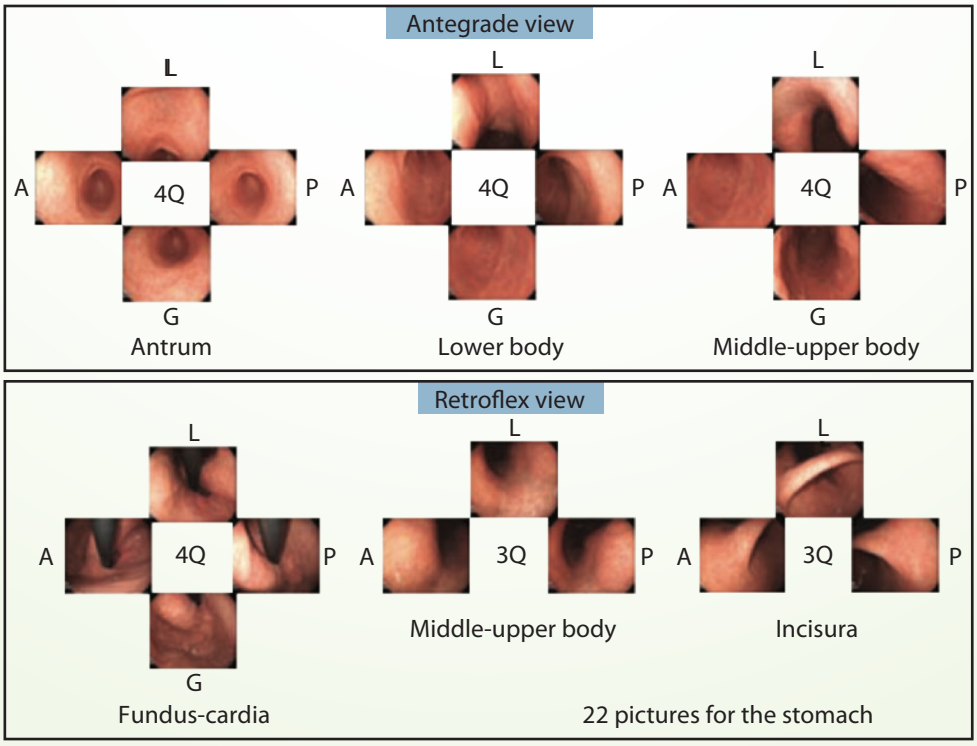
Deep learning-based AI systems developed for early gastric cancer (EGC) diagnosis were able to differentiate between gastritis and EGC, identify and delineate EGCs and determine the depth of invasion of gastric cancer with high accuracy, sensitivity, specificity and positive and negative predictive values.[7-9]

A meta-analysis of 19 studies showed a high overall AI accuracy for the diagnosis of upper GI neoplasia that was independent of the underlying condition. Although the sensitivity and specificity values in older studies were marginal compared with the standard (90%), improvements in the technology over time have increased the sensitivity and specificity values to nearly 95% in more recent studies.[10]

Polyp detection and characterization with colonoscopy and AI
Newly developed computer-aided detection (CAD) platforms have a high polyp detection rate and can locate polyps even from quickly flipped images and behind folds, noted Dr Chan. In a prospective randomized controlled study published recently, patients were randomized to standard colonoscopy (n=536) or colonoscopy with computer-aided diagnosis (n=522). The automatic polyp detection system significantly increased adenoma detection rates (ADRs; 20.3% vs 29.1%; p<0.001) and detected a significantly higher number of diminutive adenoma (102 vs 185; p<0.001) and hyperplastic polyps (52 vs 114; p<0.001).[11]

Another randomized study on colonoscopy with CAD also showed an increased ADR for colorectal polyps, in particular small polyps (≤5 mm and 6–9 mm), compared with colonoscopy without CAD.[12]

Figure. The proposed systematic screening protocol for detection of early gastric cancer[15]



Dexilant® is indicated for healing of erosive esophagitis, maintenance of healed erosive esophagitis and symptomatic non-erosive GERD

Study design[2]: A phase 1, randomized, open-label, single-center, two-period crossover study to compare the pharmacodynamic effects of single doses of Dexilant 60 mg and esomeprazole 40 mg on 24-hour intragastric pH in 44 healthy adult subjects. The primary endpoints were percentage of time intragastric pH >4 and mean pH over 24 hrs. Secondary endpoints were percentage of time intragastric pH >4 and mean pH at 0-12 hrs and at >12-24 hrs. The healthy volunteer data from this study cannot be correlated with clinical response, no clinical significance is intended or implied.

Safety Information: Contraindications: hypersensitivity to any component of the formulation, rilpivirine-containing products. Most Frequent Adverse Reactions: diarrhea, abdominal pain, nausea, upper resp tract infection, vomiting & flatulence. Serious Adverse Reactions: acute interstitial nephritis, Clostridium Difficile associated diarrhea, bone fracture, cutaneous and systemic lupus erythematosus, cyanocobalamin (Vitamin B-12) deficiency, hypomagnesemia, fundic gland polyps. For details, please consult full prescribing inform

References: **1.**Goh KL, et al. J Gastroenterol Hepatol 2014;29:1969-75. **2.**Kukulka M, et al. Clin Exp Gastroenterol 2011;4:213-20.

DEXILANT® Delayed-Release Capsules Abbreviated Prescribing Information (USPI-JUN2018-HK-SEP2018)

Active ingredient: Dexlansoprazole **Indications:** indicated in patients 12 years of age and older for healing of all grades of erosive esophagitis (EE) for up to 8 weeks; indicated in patients 12 years of age and older to maintain healing of EE and relief of heartburn. Dexilant may be used up to 6 months in adults and up to 16 weeks in patients 12-17 years of age; indicated in patients 12 years of age and older for the treatment of heartburn associated with symptomatic non-erosive gastroesophageal reflux disease (GERD) for 4 weeks. **Dosage:** Healing of Erosive esophagitis 60mg once daily up to 8 weeks, 30mg once daily for maintenance of healed EE and relief of heartburn. Symptomatic non-erosive GERD 30mg once daily for 4 weeks. **Administration:** Swallow whole. Alternatively, capsule may be opened & entire contents sprinkled in a tablespoon of applesauce. Swallow immediately without chewing. Capsule may also be opened and granules can be administered with water in an oral syringe or via a nasogastric tube (≥ 16 French). **Contraindications:** Hypersensitivity & anaphylaxis. **Special Precautions:** Gastric malignancy, acute interstitial nephritis, Increased Risk of Gastrointestinal Infections and Clostridium Difficile Associated Diarrhea, bone fracture, Cutaneous lupus erythematosus and systemic lupus erythematosus, Vitamin B-12 deficiency, hypomagnesemia, interactions with investigations for neuroendocrine tumors, interaction with methotrexate, fundic gland polyps. **Adverse Reactions:** Diarrhea, abdominal pain, nausea, upper resp tract infection, vomiting & flatulence. **Interactions:** Atazanavir, rilpivirine, digoxin, iron salts, ketoconazole, warfarin, tacrolimus & rifampin.

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C-APROM/HK/DEXI0029 (05/2022)

A retrospective analysis of the diagnostic performance of an AI-based system, EndoBRAIN, found that it could accurately detect the presence of polyps from still images produced via white-light microscopy or endocytoscopy with a high sensitivity, specificity and negative predictive value compared with endoscopist trainees, but the results of polyp characterization were comparable between the AI system and humans.[13]

Quality control

Oesophago-gastro-duodenoscopy (OGD) is a pivotal procedure in the diagnosis of upper GI lesions, but protocols from practice guidelines are often not well followed due to shortage of supervision and availability of practical tools, especially in developing countries. A meta-analysis of 10 studies (n=3,787) showed that 11.3% of upper GI cancers were missed at endoscopy within 3 years before diagnosis.[14] Therefore, a systematic screening protocol was suggested by Professor Kenshi Yao from Japan where 22 endoscopic photos should be captured in the entire stomach to reduce the missed detection rate **(Figure)**.[15]

Various AI systems have been developed to improve the OGD performance of, and assist training for, endoscopists. For example, the WISENSE system – developed by a Chinese group using the methods of deep CNNs and deep reinforcement learning – reduced the blind spot rate of OGD procedures and improved the quality of endoscopy.[16]

Future directions

The use of AI systems in detection and characterization of malignant conditions can be extended to premalignant conditions, such as intestinal metaplasia, atrophic gastritis and *Helicobacter pylori* infection. In the future, the systems may be able to generate an automated endoscopy report with relevant photographic documentation, standardized terminology and inclusion of performance measures as defined by international guidelines, commented Dr. Chan.

Conclusion

The use of AI in gastroenterology has increased efficiency and improved the quality of OGD, and it is becoming an important tool for education and training of less experienced endoscopists. However, AI cannot replace trained endoscopists as the field still requires basic endoscopy skills. Furthermore, the use of advanced imaging techniques for characterization requires thorough training to capture stable endoscopic images. Hence, the good diagnostic performances of the current AI systems for both upper and lower GI endoscopy and well-trained endoscopists are complementary in improving the quality of daily endoscopy.

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Scientific Updates

A retrospective study into etiologies and clinical outcomes of severe acute liver injury in Hong Kong (Summary of Thesis 2021)

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Introduction

Severe acute liver injury (ALI) contributes to significant morbidity and mortality and is frequently seen especially in critical illness. Early recognition, identification and treatment of the underlying cause are required for better clinical outcome. However, there have been limited studies[1-4] assessing the most common etiology of severe acute liver injury. Many of these publications involved small number of patients and their conclusions may not be applicable to our locality. This study aimed to investigate the common causes and outcomes of serum ALT ≥1000 U/L in patients admitted to a district hospital in Hong Kong.

Methods

Study design

This was a retrospective, single center cohort study conducted in Tseung Kwan O Hospital, a regional hospital in HK, between Jan 2017 – Dec 2019. Inclusion criteria included patients admitted with one or more episodes of ALT ≥1000 U/L. Exclusion criteria were (1) age under 18 years old, (2) non-ethnic-Chinese, (3) died or were discharged before evaluation of the cause of increased ALT.

Serum ALT data was identified through the Department of Pathology computerized biochemical database. Additional data were obtained from electronic medical records (ePR) for analysis including patient demographics and co-morbidities. Primary outcome was 30-day all-cause mortality from the collection date of ALT ≥1000 U/L. Secondary outcomes included length of hospital stay, need of ICU admission and liver transplantation. Data analysis was performed using SPSS version 23.0 software.

Results

There were 338 patients with one of more episodes of ALT ≥1000 U/L admitted to Tseung Kwan O Hospital between January 2017 and December 2019. A total of 313 patients were finally included in the study after excluding patients based on the exclusion criteria (12 patients died on arrival before further investigations could be made, 9 patients were non-Chinese and 4 patients were under 18 years old). Baseline demographic data and clinical characteristics were summarized in **Table**.

Etiology and outcome

The distribution of etiologies and survival outcome are shown in **Figure**. Among the 313 patients with ALT ≥ 1000 U/L, the most common causes of ALI included acute ischemia (163, 52.1%), biliary pathology (60, 19.2%), viral hepatitis (44, 14.1%) and DILI (20, 6.4%). Fourteen patients (4.5%) had no identifiable cause after reviewing the clinical history and excluding the common causes of ALI. In patients with a more extreme elevation of ALT ≥3000 U/L, ischemic hepatitis remained the leading cause (41; 70.1%), followed by viral hepatitis (9: 15.5%) and DILI (3:5.2%).

Primary outcome

The overall 30-day mortality rate was 43.1% (135 patients). Most of them (123, 91.1%) suffered from ischemic hepatitis, followed by biliary pathology (4, 3%), infiltrative liver disease (4, 3%), viral hepatitis (2, 1.5%), heat stroke (1, 0.7%) and 1 patient (0.7%) suffered from an unknown cause of acute liver injury. Among the different causes of ALI, ischemic hepatitis (OR 40, 95% CL 5.1-315.2, p<0.001) and hepatitis due to infiltrative liver disease (OR 26, 95% CI 1.8 – 367.7, p=0.002) were found to be associated with higher mortality.

Secondary outcomes

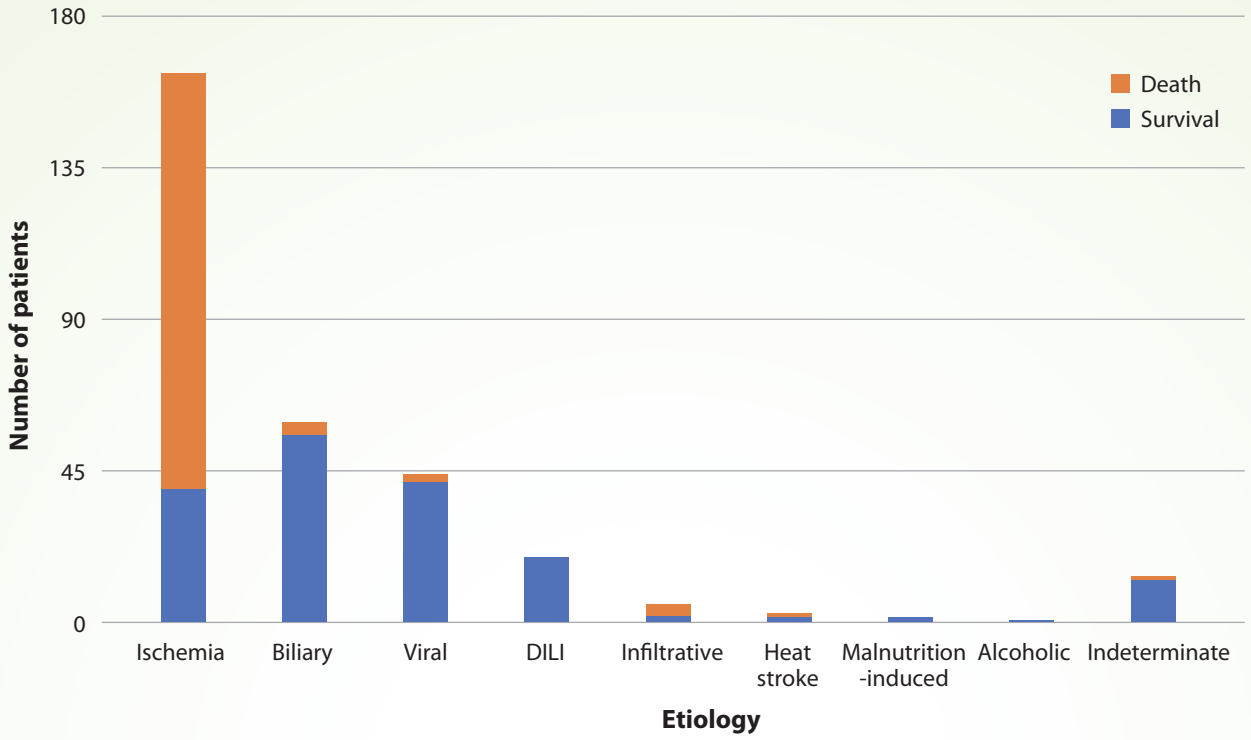
A total of 71 (22.7%) patients in our study required ICU admission. Sixty-four (90%) of them had ischemic hepatitis, followed by 2 (2.8%) patients with biliary pathology, 2 (2.8%) patients with DILI and 2 (2.8%) patients with heat stroke. Ischemic hepatitis (OR 8.4, 95% CI 1.1-65.8, p=0.043) and heat stroke (OR 26, 95% CI 1.1-604.5, p=0.042) were associated with an increased likelihood of ICU admission. The median length of stay (LOS) was 6 (3-12) days for all the patients admitted in our study. Patients who survived had a longer median LOS than those who died (8 [5-15] days vs 3 days [1-9], p<0.001). The median LOS among the top four common causes of ALI were 7 (4-10) days in the ischemic group, 7 (4-9) days in the biliary group, 5 (4-8) days in the viral hepatitis group and 9.5 (7-14) days in the DILI group. Liver transplantation was not performed in any of the patients in our study.

Table. Baseline characteristic of patients with ALT ≥1000 U/L

	Total (n=313)	Patients who survived (n= 178)	Patients who died (n=135)	p value
Demographics				
Age (y), mean +/-SD	65.4+/-18.1	58.8+/-18.9	74.1+/-12.4	<0.001
Males, n (%)	183 (58.5)	96 (53.9)	87 (64.4)	0.06
Comorbidities				
CHB	45 (14.4)	32 (18)	13 (9.6)	0.05
Other liver disease*	25 (7.9)	17 (9.5)	8 (5.9)	0.1
Liver cirrhosis	12 (3.8)	6 (3.4)	6 (4.4)	0.77
DM	86 (27.5)	38 (21.3)	48 (35.6)	0.005
HT	155 (49.5)	67 (37.6)	88 (65.2)	<0.001
CKD	60 (19.2)	19 (10.7)	41 (30.4)	<0.001
IHD	64 (20.4)	28 (15.7)	36 (26.7)	0.02
CHF	60 (19.2)	29 (16.3)	31 (23)	0.14
CVA	35 (11.2)	10 (5.6)	25 (18.5)	<0.001
COPD	20 (6.4)	8 (4.5)	12 (8.9)	0.16
Asthma	3 (1)	1 (0.6)	2 (1.5)	0.58
History of cancer	48 (15.3)	21 (11.8)	27 (20)	0.05
Presence of active cancer	23 (7.3)	7 (3.9)	16 (11.9)	0.01
Biochemistry				
Platelet (10^9/L), median (IQR)	135 (73-195)	160 (110-220)	84 (46-155)	<0.001
Serum creatinine (umol/L), median (IQR)	158 (78-381)	85 (67-140)	381 (222-527)	<0.001
Albumin (g/L), mean +/-SD	27.7+/-8.2	31.5+/-7.1	22.7+/-6.8	<0.001
Bilirubin (umol/L), median (IQR)	52 (28-99)	55 (32-100)	43 (25-97)	0.09
ALP (U/L), median (IQR)	193 (135-297)	210 (147-313)	175 (118-277)	0.017
ALT (U/L), median (IQR)	1726 (1178-2519)	1378 (1115-2171)	2043 (1405-3247)	<0.001
INR, median (IQR)	1.8 (1.2-2.7)	1.3 (1.1-1.9)	2.6 (2.0-4.0)	<0.001
pH, mean +/-SD	7.20+/-0.22	7.36+/-0.09	7.09+/-0.21	<0.001
HCO3 (mmol/L), mean +/-SD	16.1+/-7.3	20.7+/-6.6	12.8+/-5.8	<0.001

* Other liver disease included 1 chronic hepatitis C, 2 alcoholic liver disease and 25 non-alcoholic fatty liver disease
Data were expressed as mean +/-SD, median (IQR) or n (%).
CHB, chronic hepatitis B; DM diabetes mellitus; HT, hypertension; CKD, chronic kidney disease; IHD, ischemic heart disease; CHF, congestive heart failure; CVA, cardiovascular accident; COPD, chronic obstructive pulmonary disease; ALP, alkaline phosphatase; ALT, alanine aminotransferase; INR, international normalised ratio; HCO3, bicarbonate.

Figure. Distribution of causes and outcomes of patients admitted with ALT ≥1000 IU/L



Discussion

Several studies[1-4] have been published in the literature evaluating the etiologies and outcomes of severe acute liver injury but only a few studies focused on Chinese population.[3,4] To our knowledge, this is the first study evaluating the common etiologies and clinical outcomes of patients with ALT ≥1000 U/L in Hong Kong.

Our study confirmed that ischemic hepatitis was the most common cause of severe acute liver injury in Hong Kong. Over 50% of patients with ALT ≥ 1000 U/L and 70% of patient with ≥3000 U/L in our study were due to ischemic hepatitis which was consistent with findings from previous studies from other localities.[1-4] Although ischemic hepatitis is a potentially reversible condition, it is associated with high mortality and often requires ICU care. It has been reported that ischemic hepatitis was associated with >50% mortality[5] and our study showed that 75% of patient with ischemic hepatitis died during their hospital stay. The direct cause of death is usually due to the underlying illness, and not the liver injury itself. Early detection with prompt treatment aiming at correcting the underlying cause of hypoperfusion is therefore the key to achieve a better patient outcome.

The second most common cause of severe ALI was found to be biliary pathology which is contrary to the traditional teaching of differential diagnosis for ALI. Biliary obstruction is usually associated with cholestasis and less commonly presented with severe transaminitis. However, recent studies have reported biliary obstruction as being one of the most common causes of marked elevation of ALT in both in the Western and Asian regions.[1-2,4] The proposed mechanism of hepatocellular injury with biliary obstruction is due to an increase in biliary pressure resulting in retained bile salt which leads to bile salt induced hepatocyte necrosis or apoptosis.[6] Apart from biliary obstruction, 7 patients in our study with cholecystitis but without evidence of choledocholithiasis were also found to have marked elevation of ALT. One postulation is that there could be small stones passing through the biliary duct with transient biliary obstruction and biochemical abnormality. By the time of investigation, these stones had already passed, and thus no evidence of common bile duct (CBD) obstruction could be documented. Other proposed mechanisms suggested that the presence of free radical reactions in cholecystitis induces oxidative stress and liver injury.[7-8] The inflammation in the gallbladder may also cause inflammation in the nearby liver tissue, resulting in ALI.

Viral hepatitis was unsurprisingly found to be the third common cause of severe ALI and is consistent with previous studies. The type of viral hepatitis was different from the Western countries. Hepatitis B virus (HBV) infection is still prevalent in Hong Kong and was the commonest cause of severe ALI in our patients whereas hepatitis C virus (HCV) infection is more commonly found in the Western population.

Drug induced liver injury was the fourth most common cause of severe ALI in this study. It was consistently found to be one of the top common causes reported in the literature. The type of causative drug seemed to be different between Western and Asian countries. Antibiotics were shown to be the most common cause of DILI in United State and European countries whereas

herbal medicine was the most common causative agent in Asian countries.[9-10] Our study also shows a similar pattern with 9 out of 20 patients (45%) in the DILI group having a causative agent due to herbal medicine or traditional Chinese medication. Despite the overall good prognosis in most DILI patients, a small group of patients may progress to liver failure resulting in mortality. There is generally no specific treatment for DILI apart from a selective group of drugs (e.g. N-acetylcysteine for paracetamol overdose), thus early recognition is very important to prevent ongoing liver injury by the offending agent.

Conclusion

In conclusion, the common causes of severe ALI in Hong Kong included ischemic hepatitis, biliary pathology, viral hepatitis and DILI. Ischemic hepatitis was associated with high mortality thus early recognition with treatment aiming at reperfusion would be vital. Biliary pathology has always been an under-recognized cause of ALI and physicians should have a higher clinical suspicion when encounter patient with abdominal pain and ALI. Viral hepatitis B remained the leading viral cause in Hong Kong and early initiation of antiviral is recommended to prevent adverse outcome. Diagnosis and identification of the causative drug in DILI can be challenging. Obtaining a detailed drug history, in particular the use of herbal medication, is essential in Chinese population. Prospective studies with larger sample size are warranted in the future to further evaluate the clinical characteristics of each subgroup of patients.

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24th JOINT ANNUAL SCIENTIFIC MEETING 2022 (Hybrid)

Sunday, 18 September 2022 1:00pm

Venue: Level 8, Cordis Hong Kong, Mongkok

Organizing Chairman: Prof. Wai-Keung Leung

Organizers:
The Hong Kong Society of Gastroenterology
Hong Kong Society of Digestive Endoscopy
Hong Kong Society for Coloproctology
The Hong Kong Association for the Study of Liver Diseases
The Hong Kong Society of Gastrointestinal Motility
Hong Kong IBD Society

Topics	Speakers
Pharyngeal manometry and interpretation	Prof. Taher Omari (Australia)
Novel machine learning models for HCC risk prediction	Prof. Grace L.H. Wong (CUHK)
Indications and outcomes of endoscopic full thickness resections	Dr. Hon-Chi Yip (CUHK)
Management of Pancreatic Cystic Lesion in Hong Kong	Dr. Tan-To Cheung (HKU)
Intestinal USG for IBD	Prof. Torsten Kucharzik (Germany)
Total neoadjuvant therapy for rectal cancer	Dr. Henry K.M. Joeng (UCH)

Coming Soon

The Hong Kong Society of Gastroenterology Annual Scientific Meeting 2022 (Live Webinar)

Date: Thursday 7 July 2022, 7 pm

Organizing Chairperson: Professor Wai-Kay Seto

Presentation of Honorary Fellowship by President, Professor Wai-Keung Leung

Is cure of hepatitis B possible?

Professor Anna S.F. Lok
Assistant Dean for Clinical Research
Alice Lohrman Andrew Research
Professor of Hepatology
Department of Internal Medicine
University of Michigan, USA

Case Presentation: A patient with biliary obstruction

Dr. On-Pong Chiu
Associate Consultant
Department of Medicine & Geriatrics
Kwong Wah Hospital
Hong Kong

Scientific Sharing: Home Faecal Calprotectin Testing for IBD

Professor Wai-Keung Leung
Li Shu Fan Medical Foundation
Professor in Gastroenterology
Department of Medicine
The University of Hong Kong
Hong Kong

(More information will be available soon from www.hksge.org)

Major Meetings

10-11 June 2022
The International Digestive Endoscopy Network (IDEN) 2022 (Hybrid)
Organizer: International Digestive Endoscopy Network
Location: Seoul, Korea
Website: www.iden.or.kr/

16-18 June 2022
The 10th Annual Meeting of Asian Organization for Crohn's & Colitis (AOCC 2022) - Virtual
Hosted by: Asian Organization for Crohn's & Colitis (AOCC)
Location: Japan
Website: aocc-ibd.org

22-26 June 2022
International Liver Congress 2022 (EASL 2022)
Organizer: The European Association for the Study of the Liver (EASL)
Location: London, UK
Website: http://easl.eu/event/international-liver-congress-2022/

7-8 July 2022
Abdominal Imaging in Gastroenterology and Hepatology
Organizer: Falk Foundation e.V.
Location: Amsterdam, The Netherlands
Website: falkfoundation.org/en/events/default-title/abdominal-imaging-in-gastroenterology-and-hepatology-120/

1-3 September 2022
APASL Oncology 2022 - HCC: Clinical and basic research
Organizer: The Asian Pacific Association for the Study of the Liver
Location: Takamatsu, Japan
Website: apaslstc-takamatsu2022.org

3-4 September 2022
The International Digestive Disease Forum 2022 - Hybrid (IDDF 2022)
Organizer: Institute of Digestive Disease (IDD) of The Chinese University of Hong Kong (CUHK)
Location: Henry Cheng International Conference Centre, CUHK
Website: www.iddforum.com

3-4 September 2022
Multi-specialty Medical Mega Conference 2022 (Hybrid)
Organizer: Association of Private Medical Specialists of Hong Kong
Location: Cordis, Hong Kong at Langham Place, Hong Kong
Website: www.mmmc.hk

26-28 September 2022
18th World Congress for Esophageal Diseases
Organizer: The International Society for Diseases of the Esophagus
Host: Japan Esophageal Society
Location: Tokyo, Japan
Website: www.isde.net/event-4045534

8-11 October 2022
30th UEG Week (Hybrid)
Organizer: United European Gastroenterology (UEG)
Location: Vienna, Austria
Website: ueg.eu/p/202

17-21 October 2022
Asian Pacific Digestive Week (APDW 2022)
Organizers: Asian Pacific Digestive Week Federation (APDWF), Asia Pacific Association of Gastroenterology (APAGE), Asia Pacific Association for the Study of the Liver (APASL), Asian Pacific Society for Digestive Endoscopy (A-PSDE) and ISDS Asian Pacific
Location: Xian, China
Website: to be announced

21-26 October 2022
ACG 2022 Annual Scientific Meeting and Postgraduate Course - Hybrid
Organizer: American College of Gastroenterology (ACG)
Website: acgmeetings.gi.org/

4-8 November 2022
AASLD The Liver Meeting 2022 - Hybrid
Organizer: American Association for the Study of Liver Diseases (AASLD)
Location: Washington D.C., USA
Website: www.aasld.org/the-liver-meeting

1-3 December 2022
Korean Digestive Disease Week 2022 – Hybrid
Organizer: Korean Society of Gastrointestinal Endoscopy
Location: Incheon, Korea
Website: www.kddw.org

12-14 December 2022
World Congress of Gastroenterology (WCOG) 2022
Organizer: The World Gastroenterology Organization
Location: Dubai, UAE
Website: wcog2022.org

(More information is available from www.hksge.org/events)

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SCA-HK-000017

*Diosmectite is a clay of 100% natural origin.

Smart by Nature*. Powered by Science.

forlax® 10g
Macrogol 4000

Constipated?

Forlax® liberates you with softness

Forlax® 10g is indicated for symptomatic treatment of constipation in adults and children 8 years and above

Made in France

A full osmotic effect is brought about by a mixture of non-absorbable, non-metabolized polymers[1]

The effect becomes apparent within 24 to 48 hours after its administration[2]

24-48 hours

Treatment is suitable for use during pregnancy[2]

PEG is recommended as a first-line pharmacological treatment in primary care
in 2019 Hong Kong consensus statements on diagnosis and management of CIC[4]

PEG is recommended by multiple international guidelines for the treatment of CIC in adults with high quality of evidence[5-10]:

CIC = chronic idiopathic constipation. PEG = polyethylene glycol.
PRESENTATION AND FORM Powder for oral solution. Box of 10 or 20 sachets. **THERAPEUTIC INDICATION** FORLAX 10g is indicated in symptomatic treatment of constipation in adults and children aged 8 years and above. An organic disorder should have been ruled out before initiation of treatment. FORLAX® 10g should remain a temporary adjuvant treatment to appropriate lifestyle and dietary management of constipation, with a maximum 3-months treatment course in children. If symptoms persist despite associated dietary measures, an underlying cause should be suspected and treated. **DOSAGE AND ADMINISTRATION** The dosage is 1 to 2 sachets per day. The content of each sachet must be dissolved in a glass of water for oral consumption. The effect of FORLAX® becomes apparent within 24 to 48 hours after its administration. In children, treatment should not exceed 3 months. Treatment induced restoration of bowel movement will be maintained by lifestyle and dietary measures. In all cases, you must strictly comply with your doctor's prescription or your pharmacist's advice. **CONTRAINDICATIONS** Severe inflammatory bowel disease (ulcerative colitis, Crohn's disease), toxic mega colon, perforation or risk of perforation, ileus or suspicion of intestinal obstruction or symptomatic stenosis, painful abdominal syndromes of undetermined reason, and known hypersensitivity to FORLAX® or to one of the components. Due to the presence of sorbitol (traces), this medicine is contraindicated in persons who are intolerant to fructose. **DRUG INTERACTIONS AND OTHER INTERACTIONS FORLAX®**may interfere with the absorption of other drugs if administered simultaneously. Generally, it is better to take FORLAX® at least 2 hours apart from other products.**ADVERSE EFFECTS** Adverse Drug Reactions are listed under headings of frequency using the following categories: Very common (≥1/10); common (≥1/100 to <1/10); uncommon (≥1/1,000 to <1/100); rare (≥1/10,000 to <1/1,000); very rare (<1/10,000); unknown (cannot be estimated from the available data). Adult population: The undesirable effects listed below have been reported during clinical trials (including 600 adult patients) and postmarketing use. Generally, adverse reactions have been minor and transitory and have mainly concerned the gastrointestinal system: **Adverse reactions: Gastrointestinal disorders (Common)** Abdominal pain and/ or distension, Diarrhoea, Nausea **(Uncommon)** Vomiting, Urgency to defecate, Fecal incontinence **Metabolism and Nutrition Disorders (Unknown)** Electrolytes disorders (Hyponatremia, Hypokalaemia) and or dehydration, especially in elderly patients **Immune system disorders (Very rare)** Hypersensitivity reactions (Pruritus, Rash, Face oedema, Quincke oedema, Urticaria, Anaphylactic shock) **Paediatric population**: The undesirable effects listed below have been reported during clinical trials including 147 children aged from 6 months to 15 years and post-marketing use. As in adult population, adverse reactions have generally been minor and transitory and have mainly concerned the gastrointestinal system: **Adverse reactions: Gastrointestinal disorders (Common)** Abdominal pain, Diarrhoea ***(Uncommon)** Vomiting, Bloating, Nausea Immune system disorders (Unknown) Hypersensitivity reactions **STORAGE CONDITIONS AND SHELF LIFE** To be stored below 25°C, Do not exceed the expiry date plainly indicated on the pack, Keep out of reach of children **Country of origin**: France

References
1. Bouhnik Y, Neut C, Raskine L, et al. Prospective, randomized, parallel-group trial to evaluate the effects of lactulose and polyethylene glycol-4000 on colonic flora in chronic idiopathic constipation. Aliment Pharmacol Ther 2004; 19: 889–899. 2. Forlax® Hong Kong prescribing information. 3. Cleveland Clinic. Health Library. Disease & Conditions. Constipation. Available at: https://my.clevelandclinic.org/health/diseases/4059-constipation (Accessed June 08, 2021). 4. Wu JCY, Chan AOO, Cheung TK, et al. Consensus statements on diagnosis and management of chronic idiopathic constipation in adults in Hong Kong. Hong Kong Med J 2019;25:142-148. 5. Paré P, Bridges R, Champion MC, et al. Recommendations on chronic constipation (including constipation associated with irritable bowel syndrome) treatment. Can J Gastroenterol 2007;21(Suppl B):3B-22B. 6. Schmulson Wasserman M, Francisconi C, Olden K, et al. [The Latin-American Consensus on Chronic Constipation]. Gastroenterol Hepatol 2008;31:59-74. 7. Lindberg G, Hamid SS, Malfertheiner P, et al. World Gastroenterology Organisation global guideline: Constipation--a global perspective. J Clin Gastroenterol 2011;45:483-487. 8. Bove A, Bellini M, Battaglia E, et al. Consensus statement AIGO/SICCR diagnosis and treatment of chronic constipation and obstructed defecation (part II: treatment). World J Gastroenterol 2012;18:4994-5013. 9. Ford AC, Moayyedi P, Lacy BE, et al. American College of Gastroenterology monograph on the management of irritable bowel syndrome and chronic idiopathic constipation. Am J Gastroenterol 2014;109(Suppl 1):S2-26; quiz S27. 10. Shin JE, Jung HK, Lee TH, et al. Guidelines for the Diagnosis and Treatment of Chronic Functional Constipation in Korea, 2015 Revised Edition. J Neurogastroenterol Motil 2016;22:383-411.

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